Dissertation on

A CLINICAL STUDY ON 100 CASES OF NEOVASCULAR GLAUCOMA

Submitted in partial fulfillment of requirements of

M.S. OPHTHALMOLOGY

BRANCH – III

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MADRAS MEDICAL COLLEGE

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CHENNAI

MAY 2018

CERTIFICATE

This is to certify that this dissertation entitled "**A CLINICAL STUDY ON 100 CASES OF NEOVASCULAR GLAUCOMA**" is a bonafide record of the research work done by **Dr. K. SUGEPRIYA**, post graduate in Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Madras Medical College and Government General Hospital, Chennai-03, in partial fulfillment of the regulations laid downby The Tamil Nadu Dr. M.G.R. Medical University for the award of M.S. Ophthalmology Branch III, under my guidance and supervision during the academic years 2015-2018.

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I wish to express my sincere thanks to my father and mother and to all my senior post graduates and colleagues who had helped me in bringing out this study.

DECLARATION BY THE CANDIDATE

I hereby declare that this dissertation entitled "**A CLINICAL STUDY ON 100 CASES OF NEOVASCULAR GLAUCOMA**" is a bonafide and genuine research work carried out by me under the guidance of Prof.Dr.P.S.Maheswari.

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Dear Dr.K.Sugepriya,

The Institutional Ethics Committee has considered your request and approved your study titled **"A CLINICAL STUDY ON 100 CASES OF NEOVASCULAR GLAUCOMA" - NO.13012017 (III).**

The following members of Ethics Committee were present in the meeting hold on **24.01.2017** conducted at Madras Medical College, Chennai 3

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We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

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INTRODUCTION

Glaucoma is considered to be the second major cause of blindness next only to cataract. It causes irreversible blindness. It has been found to affect 66 million persons worldwide. Due to its nature to produce irreversible blindness, early detection and management is mandatory.

The meaning of the word glaucoma in Greek is “clouded”. Glaucoma can be defined as chronic progressive optic neuropathy, the salient findings being, characteristic optic disc changes with corresponding defects in visual field. Intraocular pressure may or maynot be raised. The only modifiable risk factor is the intraocular pressure.

Glaucoma can be divided into

- Congenital
- Primary and
- Secondary forms

Primary glaucomas can again be divided into primary open angle glaucoma and primary angle closure glaucoma. These have the usual salient findings of optic disc changes and visual loss along with open angles and closed angles on gonioscopy respectively. Two variants of primary open angle glaucomas are ocular hypertension and normotensive glaucoma. Some believe they are separate entities.

Ocular hypertension refers to conditions where intraocular pressure is elevated without any optic nerve head changes or changes in the visual fields.Normotensive or low tension glaucomas refer to entities that have the glaucomatous optic disc changes

and visual field defects but the intraocular pressure is never more than 21mmHg.Primary glaucomas are essentially bilateral. No association with ocular or systemic diseases are seen in primary glaucomas.

Secondary glaucomas, on the other hand, are usually unilateral (may also be bilateral), usually associated with other ocular or systemic disorders. Secondary glaucomas can again be divided into open angle and closed angle forms.Some of the causes of secondary open angle glaucoma include, pigmentary glaucoma, pseudoexfoliation glaucoma, steroid induced glaucoma, lens induced like phacolyticand lens particle glaucoma, ghost cell glaucoma, haemolytic glaucoma, UGH syndrome (uveitis, glaucoma and hyphaema), Posner schlossmannsyndrome, Fuchs heterochromiciridocyclitis etc.

Secondary angle closure glaucoma causes include, Iridocorneal endothelial syndromes, posterior polymorphous dystrophy, fibrous ingrowth, penetrating keratoplasty, aniridia, ciliary block or malignant glaucoma, intraocular tumors, nanopththalmos, suprachoroidal haemorrhage and ciliochoroidal effusions, retrolental fibroplasia, neovascular glaucoma etc.

Neovascular glaucoma is a secondary glaucoma that has a potential to cause absolute glaucoma but may be prevented if the etiology identified at the right time and given a timely intervention. It may present at any stage, can present as open or closed angles. This study is about a detailed analytical study on 100 cases of neovascular glaucoma.

ANATOMY

AQUEOUS HUMOR:

Aqueous humoris a relatively protein free and cell free fluid that plays an important role in maintaining the ocular rigidity and intraocular pressure. It is formed continuously and drained. Aqueous is secreted by the non-pigmented epithelium of the ciliary process and drained by the angle of anterior chamber. It plays a pivotal role in providing nutrition to the avascular structures like cornea and lens. It not only provides nutrition but also clears the metabolites and other toxic substances. Since it is optically clear, there is no hindrance to a clear vision.

The aqueous humor flow rate is around 2.7 microlitres per minute and the volume of aqueous is 0.3 ml. It is formed from the non-pigmented epithelium of the ciliary process, into the posterior chamber, then passes through the pupil into the anterior chamber and is finally drained via 2 pathways:

- Trabecular or conventional outflow
- Uveoscleral or unconventional pathway

The two pathways of the aqueous outflow are depicted in Figure 1. The trabecular outflow occurs through the trabecular meshwork into the Schlemms canal and finally drained into the collector channels and episcleral veins. The uveoscleral outflow through the face of ciliary muscle is also depicted.

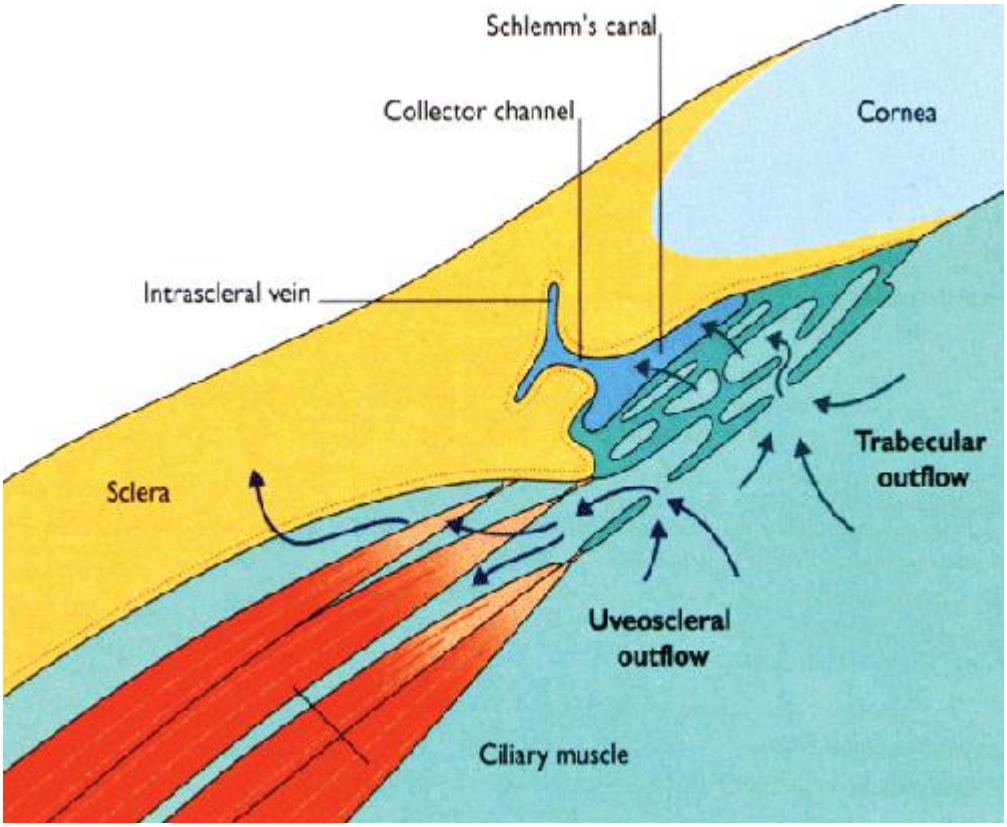


Figure 1: Diagram Showing the Aqueous Outflow

Mechanisms of aqueous production:

- Ultrafiltration
- Active secretion
- Diffusion

Diffusion:

- contributes to 10%
- lipid soluble substances are transported
- proportional to a concentration gradient across the membrane

Ultrafiltration:

- contributes to 20% of aqueous production
- transport water and water soluble substances

- Occur in response to an osmotic gradient or hydrostatic pressure
- These processes are passive and do not require energy

Active secretion:

- Na-K transport pump is the major mechanism of aqueous production
- contributes to 70%
- It is against the concentration gradient and requires energy

Composition of aqueous humor:

- Water – 99.9%
- Proteins – less than 1% of plasma proteins
- Glucose – 75% of plasma
- Sodium – similar as in plasma
- Bicarbonate ion – more in posterior chamber than anterior chamber
- Chloride more than in plasma and phosphate less than that in plasma
- Ascorbate – in very high levels to protect against UV induced oxidative stress

Blood Aqueous Barrier:

In the ciliary body barriers include the tight junctions between the non-pigmented epithelial cells and in the iris it includes the tight junctionsof endothelium of iris blood vessels. This blood aqueous barrier is responsible for the difference in composition between the plasma and aqueous.[6]

Factors affecting aqueous production:

- Diurnal variation
- Age and Sex

- Intraocular pressure / pseudofacility
- Blood flow to ciliary body
- Neural control
- Hormonal factors

Drainage of aqueous:

Conventional outflow:

Contributes to 80% of outflow and is through the angle of the anterior chamber.[6] Structures in the angle from anterior to posterior include:

- Schwalbe's line
- Trabecular meshwork
- Scleral spur
- Ciliary body band

Schwalbe's line marks the posterior end of Descements membrane and is the anterior most structure in the angle. It is seen as a white line in goniscopy. The visualisation is facilitated by using a corneal wedge in gonioscopy.

Trabecular meshwork is composed of an anterior non-pigmented and a posterior pigmented portion. It appears as a brown line in gonioscopy. It is triangular in meridional section, with apex at schwalbe's and base at scleral spur. Histologically it is divided from deep to superficial, as shown in Figure 2, as:

- Uveal meshwork
- Corneoscleral meshwork
- Juxtacanalicular tissue

Uveal meshwork consists of openings of size 25 to 75 microns – has the least resistance to aqueous flow.

Corneoscleral meshwork has openings of 5 to 50 microns – gives moderate resistance to aqueous flow.

Juxtacanalicular tissue – offers the maximum resistance to aqueous flow

Scleral spur is the posterior projection of sclera sulcus. It is made of collagen type 3 and 1. It is seen as white line in goniscopy.

Ciliary body band appears as grey or brown band in goniscopy. The anterior part of ciliary body forms this. In myopes, the ciliary band is wider.[2]

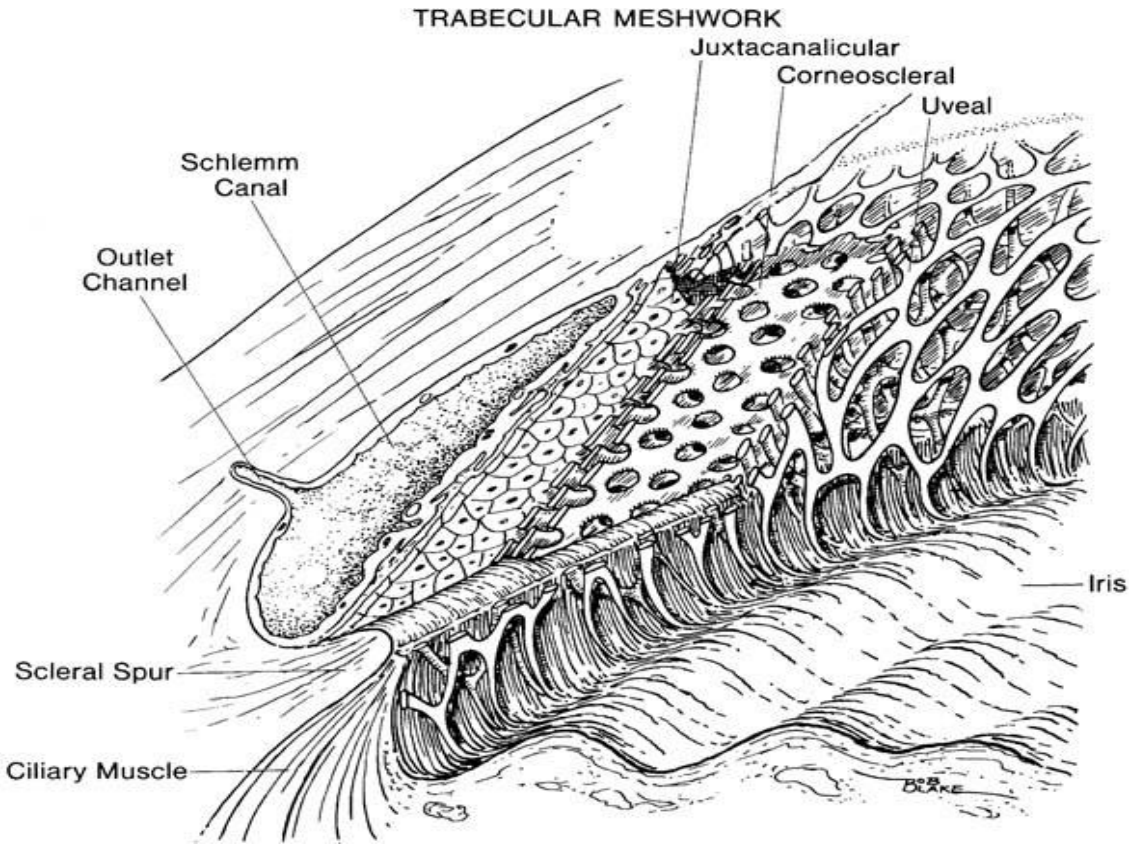


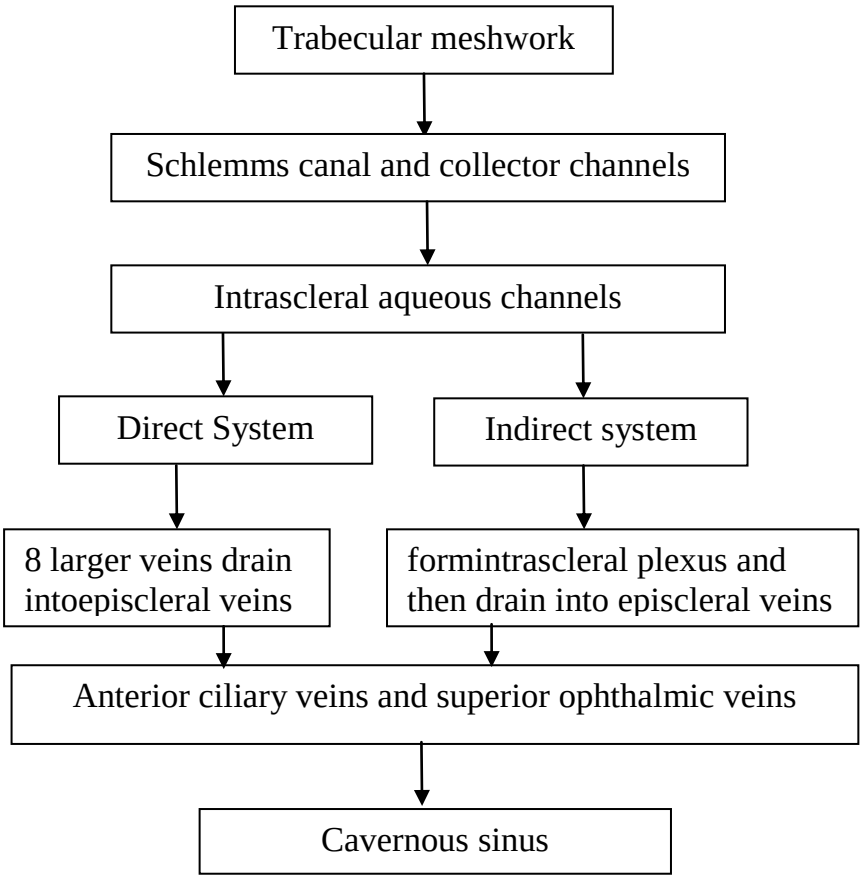
Figure 2: Diagram Showing the Parts of Trabecular Meshwork

Uveoscleral Outflow:

Contributes to 10 to 20% of aqueous outflow[(2)]

- Aqueous passes through the root of iris, between the ciliary muscle fibres and then into the suprachoroidal space
- The outflow through this route is independent of intraocular pressure
- Matrix metallo proteinase 1 enzyme plays a major role in this pathway
- Miotics will cause constriction of ciliary muscles and the flow through this route is decreased

Final Pathway:
Conventional outflow:



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In contrast, uveoscleral outflow ends by draining from suprachoroidal space into venous circulation of ciliary body, choroid and sclera

EXAMINATION OF A GLAUCOMA CASE

- A detailed ocular and systemic history
- Visual acuity
- Intraocular pressure measurement
- Central corneal thickness measurement
- Detailed Anterior segment examination with slitlamp
- ONH examination with +90D lens
- Gonioscopy
- Visual fields

History:

Detailed ocular history may help in assessing the cause in cases of secondary glaucomas:

- Previous treatment history is important
- Systemic conditions history is necessary in planning anti glaucoma medications and surgery

Visual acuity:

- Best corrected visual acuity along with near vision correction has to be noted

Intraocular pressure measurement:

- Goldmann applanation tonometer is the gold standard method
    - It is considered gold standard because the values are reproducible with that of direct manometry readings[(2)]

- Figure 3 shows the Goldmann Applanation tonometer
- It consists of a double prism mounted on a slit lamp
- Eyes are stained with flourescin and placed in slit lamp
- The applanating unit is advanced till it touches the cornea
- Two semicircles are formed and the dial has to be rotated till the inner margins of both the semicircles coincide.
- The value obtained multiplied by 10 gives the IOP in mmHg.

Principle of Goldmann Applanation Tonometer:

- Based on ImbertFicks law
- Force applied on a sphere(W) is equal to pressure inside a sphere(P) multiplied by area of applanation(A)
- W = PA
- Since the cornea is not a perfect sphere, surface tension(S) and ocular rigidity(R) interfers with the readings
- W + S = PA + R
- It is said that when the area of applanation is 3.06mm, these two variables cancel out each other and the force applied is equal to the intraocular pressure.(W = P)
- Since the volume of aqueous displaced by this method is very low – approx. 0.5 ml, the intraocular pressure measurement is not affected by ocular rigidity as in schiotz tonometer.

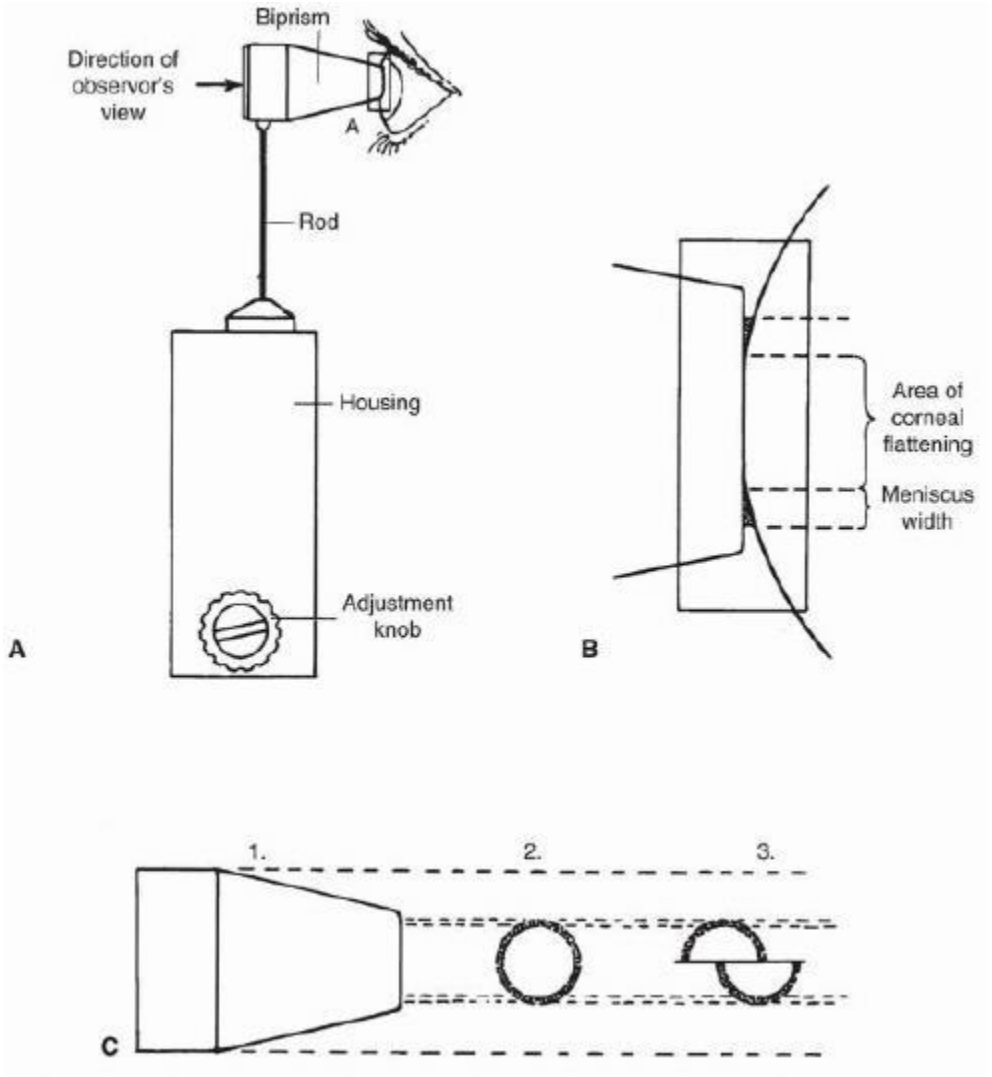


Figure 3:Diagram Showing the Goldmann Applanation Tonometer

CLASSIFICATION OF TONOMETERS:

- Applanation – prototype is GAT
- Indentation – prototype is Schiotz tonometer

Applanation tonometers are further classified into

Variable force tonometers

- Goldmann applanation tonometer
- Perkins tonometer
- Draeger tonometer

- Mackay marg tonometer
- Tonopen
- Pneumotonometer

Variable area tonometers:

- Maklakov tonometer
- Applanometer
- Tonomat
- Barraquer tonometer

Perkins tonometer:

- Similar to GAT
- Portable and handheld
- Used in children and in recumbent position

Mackay marg tonometer:

- Has a plunger with a diameter 1.5mm and surrounding sleeve of 3mm[(4)]
- Force required to keep the plate flush with the sleeve is electronically monitored and recorded in a paper strip
- Useful in scarred and irregular corneas

Pneumotonometer:

- Has a sensing nozzle whose apex touches the cornea
- Connected to a central chamber containing pressurised air
- Pneumatic to electronic transducer converts the air pressure into electronic readings and IOP is measured

Tonopen:

- Similar principle as mackay marg
- Portable and hand held
- Used in children, ward rounds and community fair

Maklakov tonometer:

- It is a variable area tonometer
- Consists of wire holder into which a flat bottom weight, from 5 to 15g, is inserted
- Surface coated with dye and placed on the cornea
- Area of missing dye is then measured and IOP calibrated

NON CONTACT TONOMETERS:

- Introduced by Grolman[4]
- Has alignment system, pneumatic system and optoelectric monitoring system
- A puff of room air is generated and applanates the cornea
- The light reflected is at maximum intensity when the cornea is applanated
- The time taken for the light reflected to reach its maximum intensity is measured and IOP calibrated
- Pulsair is a portable NCT

SCHIOTZ TONOMETER(Figure 4):

- Indentation tonometery

- Known weight is placed on the cornea and the deformation is measured
- Cornea anesthetised and patient placed in supine position
- Instrument placed on the cornea and the needle moves, the reading is noted
- Friedenwald conversion tables are used
- As the amount of aqueous displaced is large, conversion tables are required to calibrate IOP
- Useful tool in screening patients

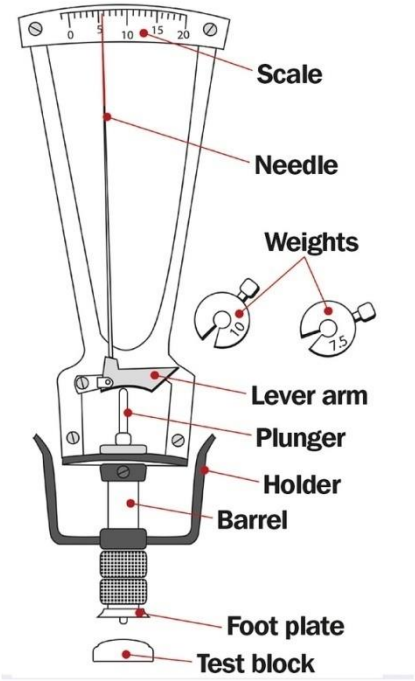


Figure 4: Diagram Showing the Schiotz Tonometer

REBOUND TONOMETER:

- Hand held and portable
- Consists of a probe that hits the cornea and come back

- The time taken for the probe to return back is faster in low IOP and slower in high IOP
- Useful tool in children,
- No need for topical anesthesia

Other types of tonometers include:

- Dynamic contour tonometer
- Ocular response analyser
- Transpalpebral tonometer

CENTRAL CORNEAL THICKNESS MEASUREMENT:

- Measured with a ultrasonic pachymeter
- Has an influence on GAT values
- GAT Overestimates in case of thicker corneas and underestimates in case of thinner corneas
- Normal CCT – 520 to 550 microns
- For each 10microns difference, IOP has to be corrected by 0.7 mmHg

Detailed anterior segment evaluation

- Using slitlamp biomicroscopy, detailed anterior segment evaluation is done
- Specific clues to the etiology has to be looked for
- Krukenberg spindle on the back of cornea, pseudoexfoliation on pupillary border and lens capsule, atrophic patches in iris, neovascularisation of iris, transillumination defects of iris, ectropion uveae, etc should not be missed and documented

ONH EVALUATION USING +90D LENS:

- Optic nerve head examination is the most important part of evaluation

- Cup disc ratio both vertical and horizontal has to be recorded (Figure 5)
- NRR thinning in poles and other quadrants has to be noted
- Vessel changes like baring of circumlinear vessels, nasalisation, bayonetting, overpass vessels has to looked for.
- Laminar dot sign and peripapillary atrophy has to be recorded
- RNFL defects has to be looked for in red free filter (Figure 6)[4][2]

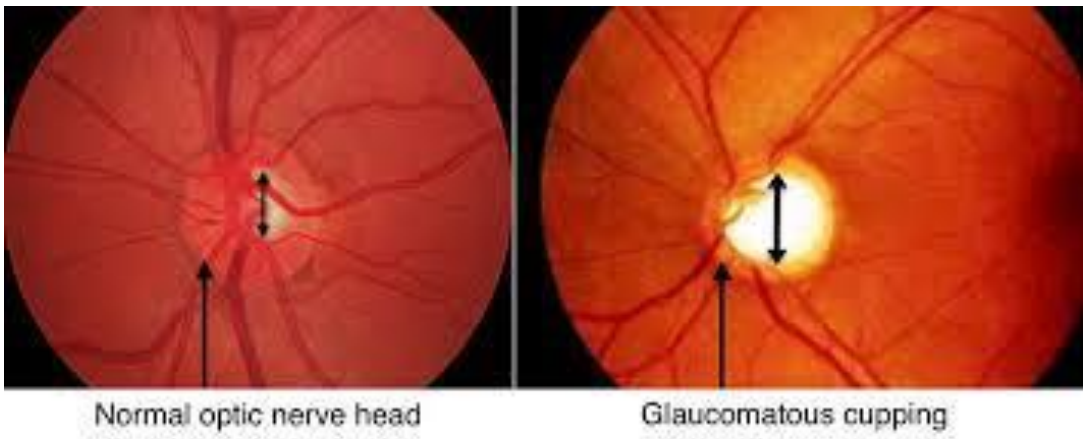


Figure 5: Diagram Showing the Normal and Glaucomatous Optic Nerve Head

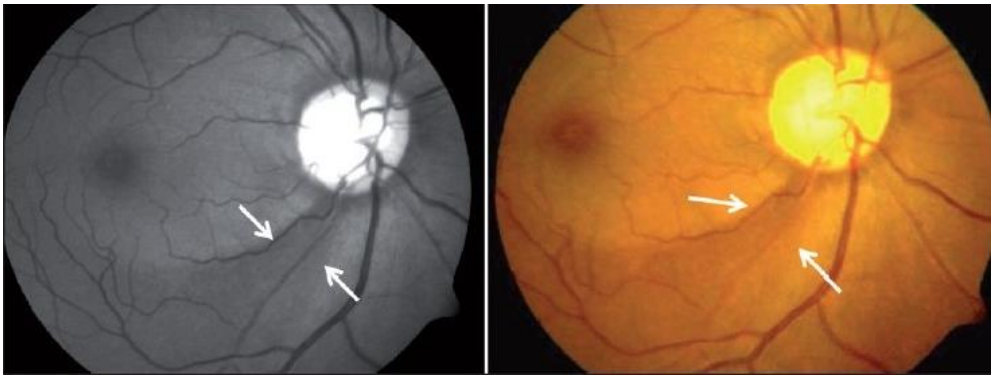


Figure 6:Diagram Showing the Retinal Nerve Fibre Layer Defects in Red Free Filter

GONIOSCOPY:

- Coined by Trantas in 1901[1]
- Evaluation of anterior chamber angle is done by gonioscopy

Principle (Figure 7):

- Based on the principle of total internal reflection
- When angle of incidence is larger than the critical angle, all the rays undergo total internal reflection and no refraction occurs[1][4]
- The cornea – air interface is such that, light from angle undergoes total internal reflection and cannot be visualised
- This is eliminated by placing a goniolens and the angle of incidence is changed, thus enabling the view of anterior chamber angle.

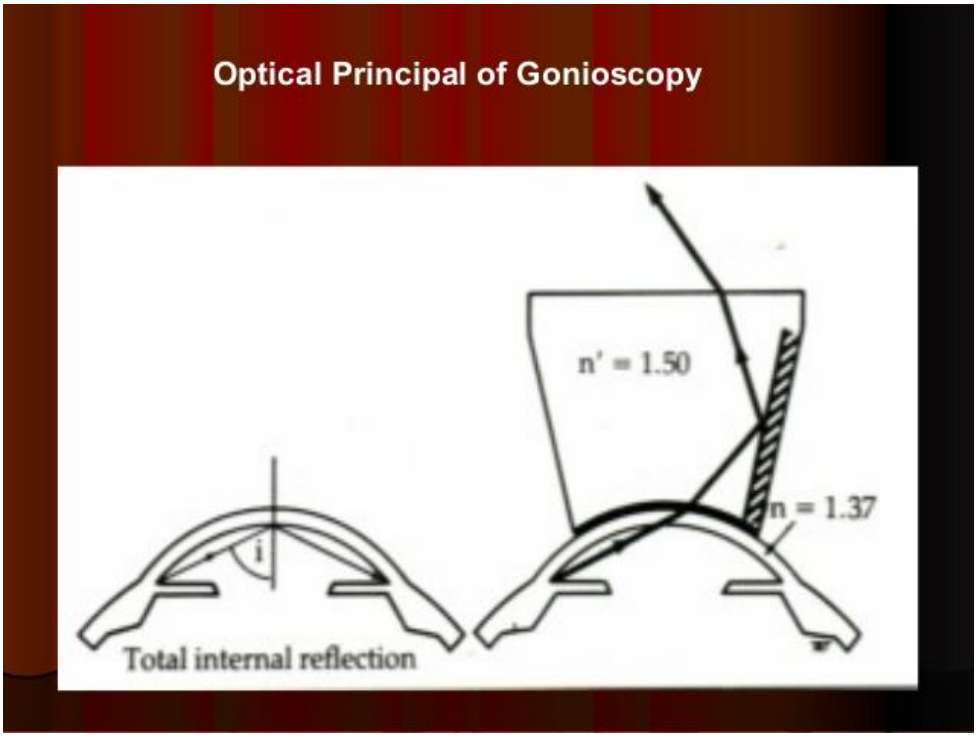


Figure 7: Diagram Showing the Principle of Gonioscopy

Gonioscopy is of two types:

- Direct
- Indirect

DIRECT GONIOSCOPY:

- Done with patient in supine position

- Uses direct goniolenses(Figure 8)
- Some of the direct goniolenses are Barkan, Koeppe and surgical goniolenses like Thorpe and Swan Jacob
- With these entire angle visualisation is possible
- Can be used to compare angles of both eyes simultaneously
- Requires a separate source of illumination and handheld microscope
- Can be used for surgical procedures like goniotomy

INDIRECT GONIOSCOPY:

- Done using indirect goniolenses(Figure 9)
- Has a mirror that reflects the light, hence the quadrant of the angle visualised is opposite to that of the mirror
- Requires a coupling fluid usually viscoelastic substance
- Examples are goldmann single mirror and three mirror, Allen Thorpe gonioprism.
- Indentation goniolenses are such that their radius of curvature is smaller than that of cornea, hence can be used to indent the cornea(Figure 10)
- These do not require coupling agent
- This indentation method is used to differentiate an appositional angle closure from synechial angle closure
- Examples of indentation goniolenses are Zeiss, Posner and Sussmann goniolens

Figure 8: Diagram showing Direct Goniolenses

Figure 9: Diagram showing Golmann three mirror indirect goniolens

Figure 10: Diagram showing Zeiss four mirror Indentation goniolens

Structures seen in gonioscopy from posterior to anterior (Figure 11):

- Root of iris
- Ciliary body band
- Sclera spur
- Trabecular meshwork
- Schwalbes line

Other structures to be looked for and differentiated include:

- Normal vs new blood vessels
- If blood is present in schlemms canal, it may be visible.
- Iris processes vs peripheral anterior synechiae
- Pigmented schwalbes vs Sampolesis line
- Specific configuration of iris like double hump sign or concave configuration

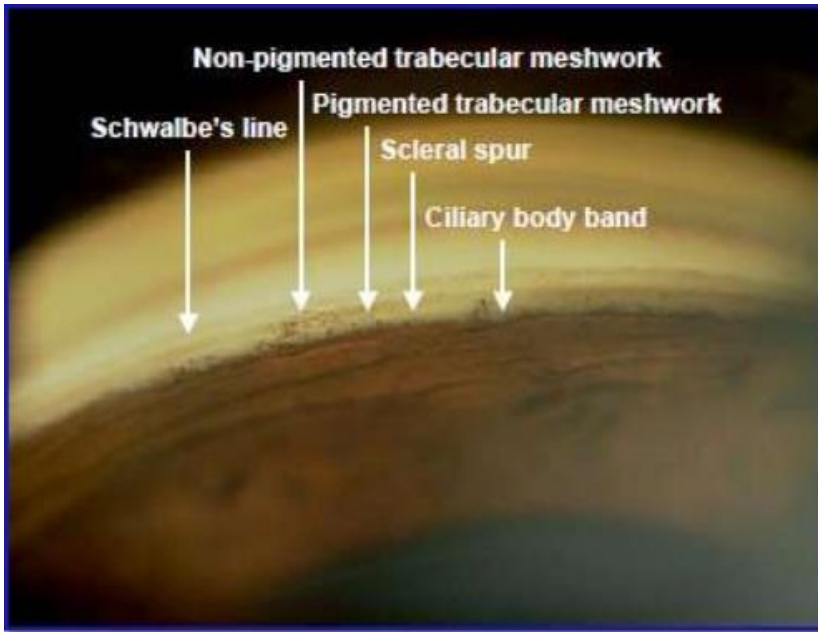


Figure 11:Diagram Showing the Normal Angle Structures

GRADING OF ANTERIOR CHAMBER ANGLE:

SHAFFERS GRADING:

- This is used routinely
- Based on angle between iris and trabecular meshwork
- Modified schaffers is based on the structures visible gonioscopically

Angle grading:

- Grade 0 : closed angle
- Grade S : slit like angle, no structures visible, <10 degrees
- Grade 1 : extremely narrow, up to schwalbe's line seen, 10 degrees
- Grade 2 : moderately narrow, functional trabecular meshwork seen, 20 degrees
- Grade 3 : non occludable , up to scleral spur seen, 30 degrees
- Grade 4 : non occludable , up to ciliary body band seen, 35 – 40 degrees

Other grading systems include:

Scheie grading:

- It is the reverse of schaffers grading

Spaeth grading:

- Uses the following components
- Level of iris root insertion
- Angular width of angle recess
- Configuration of iris
- Pigmentation of trabecular meshwork

Van Herick grading:

- It is a slit lamp method of grading

- Compares the peripheral anterior chamber depth with the corneal thickness

Table 1: Table showing Van Herick Grading of anterior chamber

ANGLE GRADING	Peripheral anterior chamber depth vs corneal thickness
Grade 4	>/= 1
Grade 3	1/4- 1/2
Grade 2	=1/4
Grade 1	<1/4

VISUAL FIELD ANALYSIS:

May be of two types:

- Kinetic perimetry
- Static perimetry

Kinetic perimetry:

- Technique where the stimulus is of constant size and intensity, and stimulus is moved from a non seeing to seeing area[(5)]
- Examples include Bjerrum screening
- Is not very accurate for glaucomatous field documentation

Static perimetry:

- Automated perimeters are included in this
- Here the stimulus is presented at various points to measure the retinal sensitivity in that point
- The stimulus size and intensity can be varied
- Examples include Octopus perimeter and Humphrey visual field analyser

Visual field defects in glaucoma (Figure 12):

- Paracentral scotoma

- Siedel scotoma
- Arcuate scotoma
- Double arcuate scotoma
- Nasal step
- Temporal step
- Tubular vision or temporal wedge of vision

Glaucomatous visual field defects can be differentiated from neurological defects in that the glaucomatous defects:

- Respect the horizontal meridian
- Are mostly incongruous and
- Are Relative field defects

Other newer types of perimeters include:

- Short Wavelength Automated Perimetry (SWAP)
- Frequency Doubled Perimetry
- High Pass Resolution Perimetry
- Flicker Perimetry
- Microperimetry

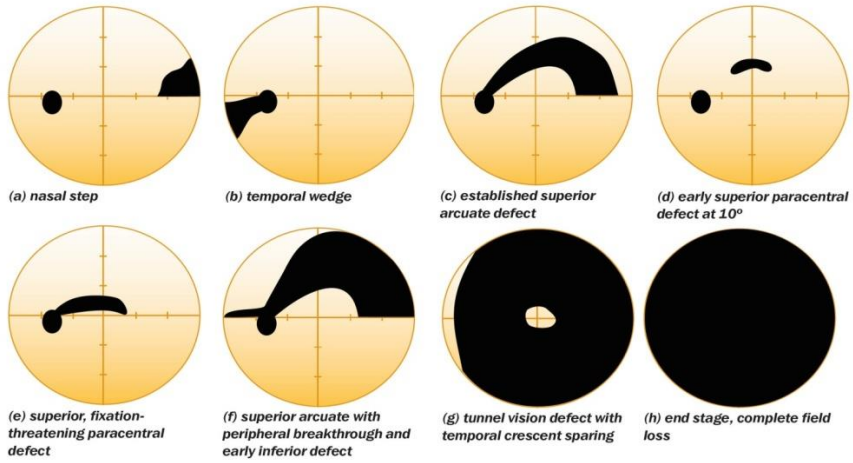


FIGURE 12: Diagram Showing the Visual Field Defects in Glaucoma

NEOVASCULAR GLAUCOMA

Neovascular glaucoma is a secondary glaucoma, due to fibrovascular membrane over either the surface of iris and/or of the angle, secondary to ocular ischemia

Other names include:

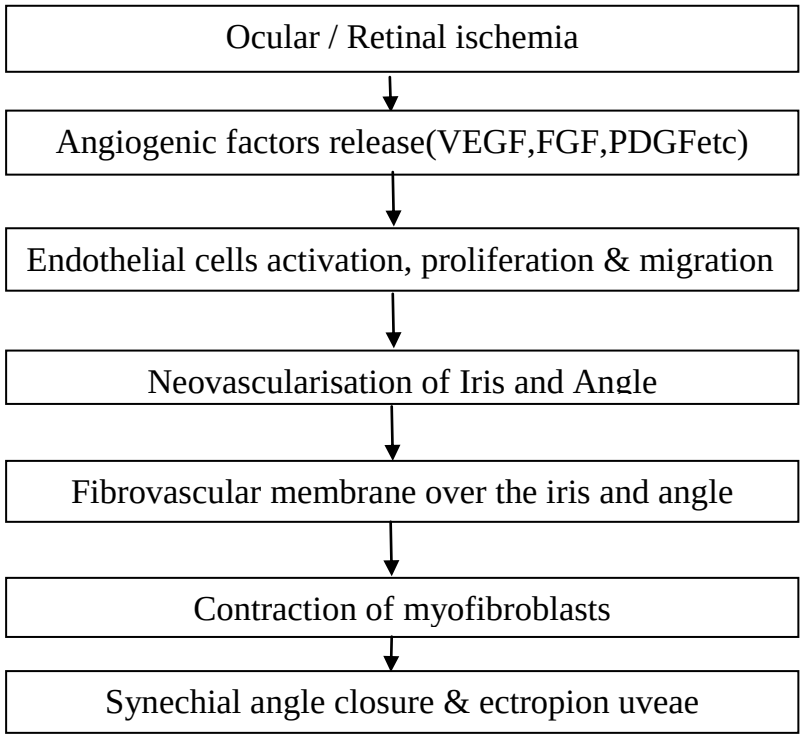
- Thrombotic glaucoma
- Hemorrhagic glaucoma
- Rubeotic glaucoma
- Congestive glaucoma

HISTORY:

First decribed in 1866 following central retinal vein occlusion

- In 1906, Coats highlighted new vessels on iris[(1)]
- Nettleship and Salus – associated diabetes and neovascular glaucoma
- Weiss and colleagues – coined Neovascular glaucoma

PATHOGENESIS:



LENS VITREOUS BARRIER (Figure 13):

- Vitreous and lens – effective in preventing angiogenic factors diffusion from posterior segment[2][4]
- Also contain high levels of anti angiogenic factors like Pigment Epithelium Derived Factor

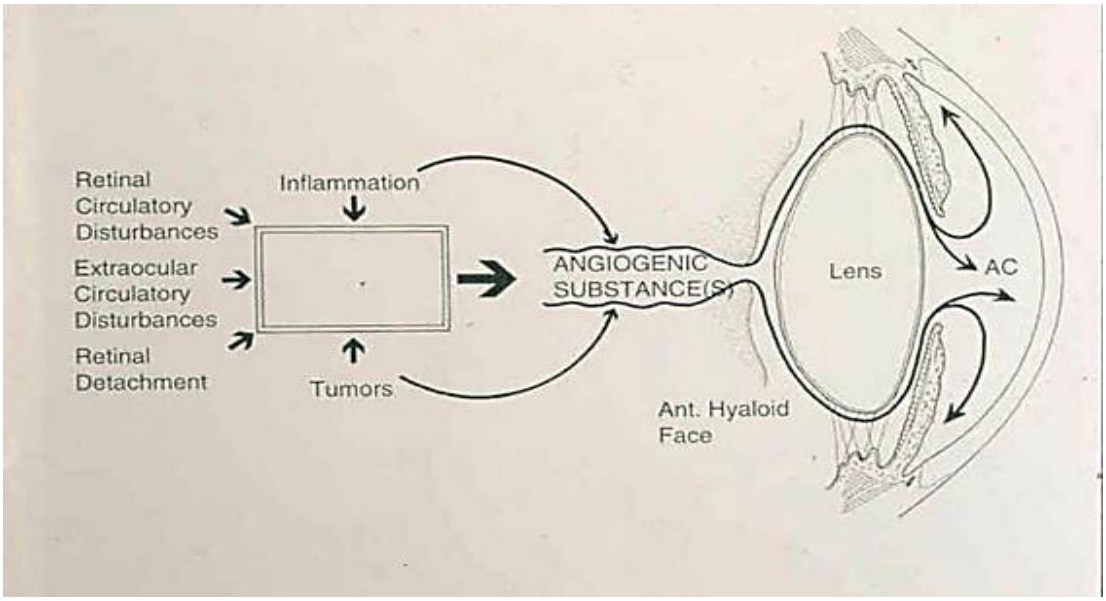


Figure 13: Diagram Showing Vitreous and Lens Barrier

ETIOLOGY:

- OCULAR VASCULAR DISEASES:
    - Central retinal vein occlusion
    - Diabetic retinopathy
    - Central retinal artery occlusion
    - Branch retinal vein occlusion
    - Branch retinal artery occlusion
    - Sickle cell retinopathy
    - Sturge weber syndrome with choroidal hemangioma

- EXTRA OCULAR CAUSES:
    - Carotid artery disease
    - Ocular ischemia
    - Carotico cavernous fistula
    - Giant cell arteritis
    - Takayasu arteritis
- TRAUMA
- OCULAR NEOPLASMS:
    - Malignant melanoma
    - Retinoblastma
- OCULAR INFLAMMATORY DISEASES :
    - Chronic uveitis
- MISCELLANEOUS:
    - Essential Iris atrophy
    - Neurofibramatosis
    - Retinal detachment

Diabetic Retinopathy:

- Contribute to one third of cases of NVG
- Usually in PDR, but also in NPDR if capillary non perfusion areas are large
- Rubeosisiridisis more common than NVG
- Higher incidence following vitrectomy and lensectomy
- Silicone oil may act as barrier and reduces occurrence of anterior segment neovascularisation

Central Retinal Vein Occlusion:

- 40% of ischemic CRVO give rise to Neovascular glaucoma
- Large areas of capillary non perfusion is an important predictive risk factor
- Can occur between 2 weeks to 2 years; usually after 3 months, hence the name – 100 day glaucoma
- Pre existing primary open angle glaucoma is a risk factor for CRVO and subsequent neovascular glaucoma

Carotid Occlusive Disease:

- Ranks as a third common cause
- Variable course
- In severe ischemia – low IOP due to low perfusion to ciliary body
- Dramatic increase occurs after surgical treatment of occlusive disease

Stages of Neovascular Glaucoma:

- Pre rubeotic stage
- Rubeotic stage
- Secondary open angle stage
- Secondary angle closure stage
- Absolute glaucoma

Rubeotic Stage (Pre glaucoma stage)

- Charecterised by iris neovascularisation with normal IOP
- New vessels in iris (Figure 14):
    - Fine tufts in arborizing pattern
    - First at pupillary margin

- Also, in margins of peripheral iridectomy

- Gonioscopy show open angles and new vessels in angle may be present

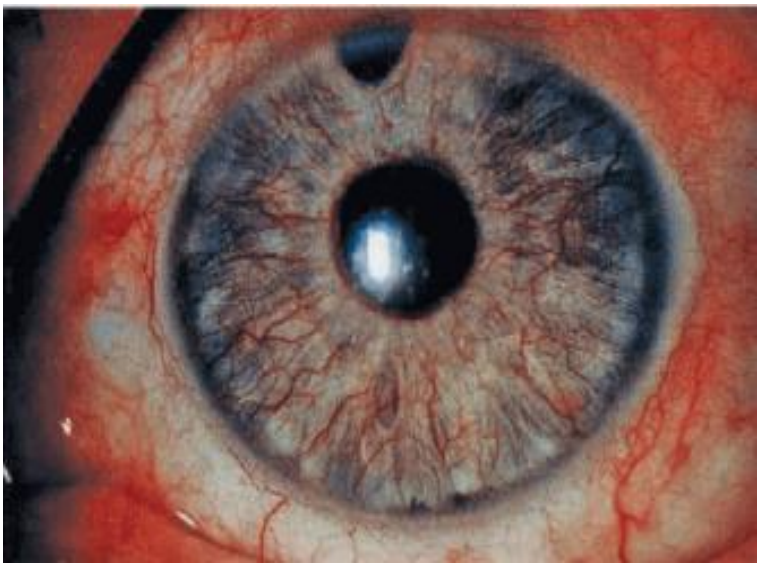


Figure 14: Diagram Showing Iris Neovascularisation

Table 2: Table Showing Difference Between Normal and New Vessels in Iris

Normal Iris Vasculature	**New Vessels in Iris**
Situated in deeper stroma	Superficially located
Radial	Arborizing
Donot leak in FA	Leak in FA

Secondary Open Angle Glaucoma Stage:

- Occlusion of trabecular meshwork by inflammatory cells and fibrovascular membrane
- Acute onset of raised IOP
- Signs:
    - Circum corneal congestion
    - Corneal edema
    - Anterior chamber – inflammatory reaction, hyphaema

- Gonioscopy : open angles with neovascularisation (Figure 15)

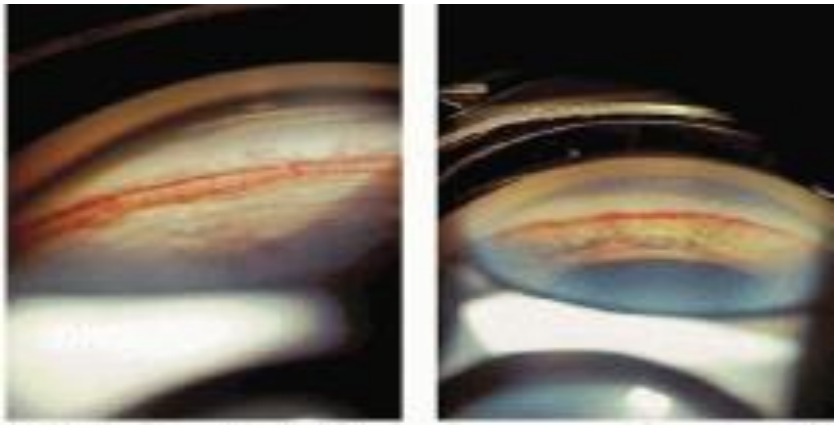


Figure 15: Diagram Showing Neovascularisation of Angle

Table 3: Table Showing Differences between Normal and New Vessels in Angle

Normal vessels in angle	**Neovascularisation of angle**
Broad, shorter segment	Fine network
Do not cross scleral spur	Cross anterior to scleral spur
Do not arborize	Arborize over trabecular meshwork

Secondary Angle Closure Stage:

- Contraction of fibrovascular membrane leads to synechial closure of angle[(4)(2)]
- Posterior pigment layer of iris is pulled to anterior surface known as Ectropion Uveae
- Signs (Figure 16):
    - Circum corneal congestion
    - Corneal edema
    - Pupil: fixed , not reacting to light
    - Neovasularisation of iris
    - Ectropion uveae

- Anterior chamber – inflammation , hyphaema
- Gonioscopy – closed angles

Figure 16: Diagram Showing Angle Closure Stage of Neovascular Glaucoma

Absolute stage:

- Intractable pain
- Very high intraocular pressure
- No perception of light

DIFFERENTIAL DIAGNOSIS:

Open angle stage:

- Glaucoma secondary to uveitis
- Fuchs heterochromic iridocyclitis

Angle closure stage:

- Acute angle closure glaucoma attack
- ICE syndromes

MANAGEMENT:

GOALS:

- Identify the cause of ischemia
- Control ischemia and decrease angiogenic factors

- Decrease IOP
- Control painful blind eye

Medical Management:

- Topical 1% atropine: to stabilise blood aqueous barrier and to relieve pain
- Topical steroids – to control anterior chamber inflammation
- IOP lowering agents:
    - Parentral hyperosmotic agents for rapid reduction of IOP – 20%Mannitol – 1-2g/kg body weight, if there is no systemic contraindication
    - Oral carbonic anhydrase inhibitors like Tab. Acetozolamide 250mg QID or 500mg BD – contraindicated in renal faiure
    - Topical anti glaucoma medications – beta blockers like 0.5% Timolol, Carbonic anhydrase inhibitors like 2% Dorzolamide or 1% Brinzolamide, Alpha agonists like 0.2% Brimonidine
    - Topical prostaglandin analogues and parasympthomimetics like pilocarine are contraindicated in neovascular glaucoma

LASERS:

Pan Retinal Photocoagulation:

- CRVOS study – if NVI > 2clockhours or NVA present – PRP to be done even in presence of normal IOP[(5)]
- PRP given prior to trabeculectomy increases the success rate
- May even reverse IOP elevation in open angle stage

Procedure:

- Topical anesthesia applied, patient made to sit in slit lamp
- Volk Quadrispheric lenses are used for PRP
- Settings are : 200 mW power, 200 milliseconds duration, 100 to 200 micron spotsize
- Lasers are given in around 3 settings – 500 burns per sitting
- Laser used is Double frequency Nd:Yag laser

Grading of burns:

- Light – barely visible blanching
- Mild – faint white retinal burn
- Moderate – opaque dirty white burn
- Heavy – dense white burn

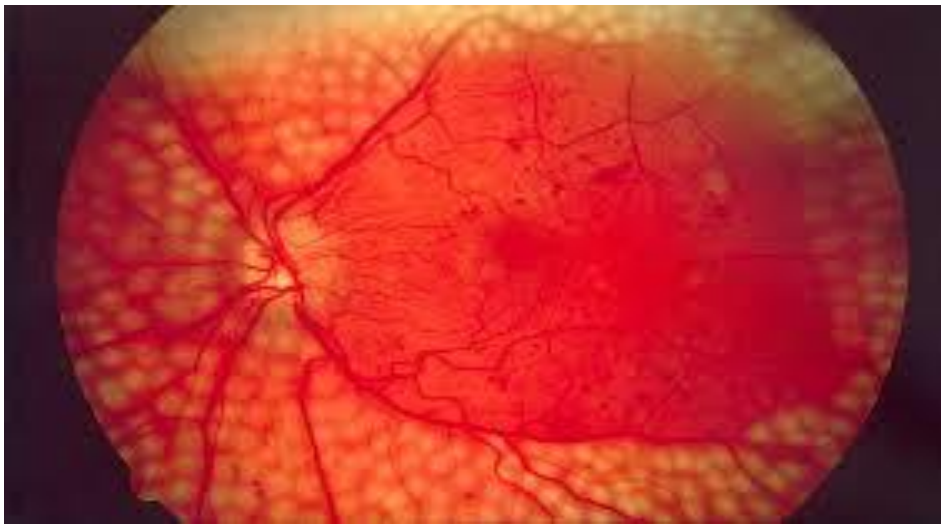


Figure 17: Diagram Showing Pan Retinal Photocoagulation Burns

Goniophotocoagulation (Figure 18):

- Low energy laser applied to the tufts of new vessels across the scleral spur
- Useful adjunct to PRP

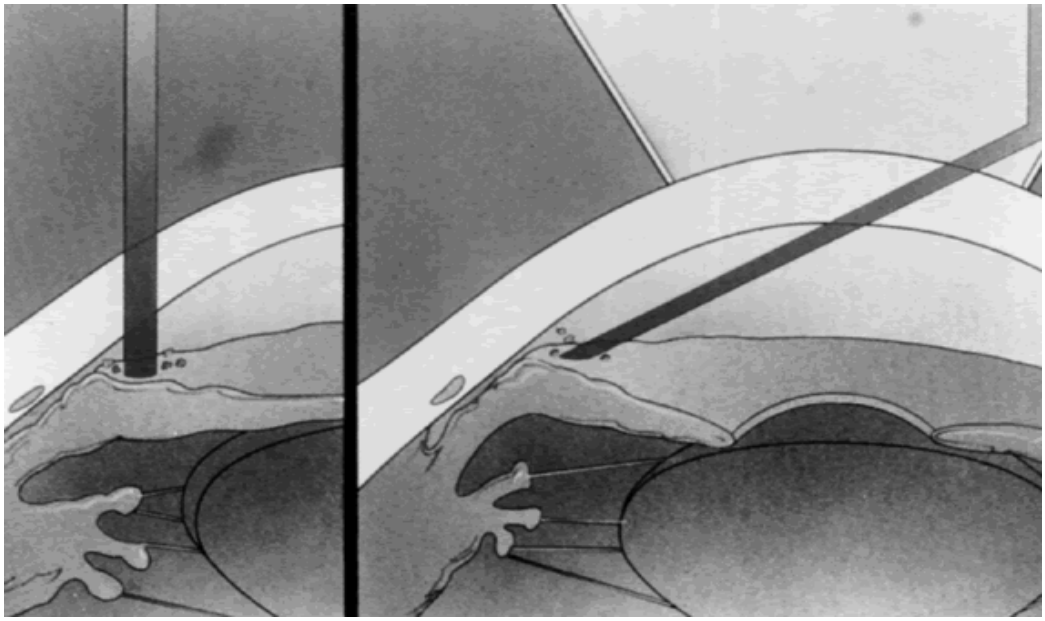


Figure 18: Diagram Showing Goniophotocoagulation

Anterior Retinal Cryotherapy:

- When media is cloudy , anterior retinal cryotherapy can be done to decrease the retinal ischemia
- 40 thaws , 10 thaws in each quadrant usually applied

Intravitreal Anti VEGF:

- Causes resolution of iris and retinal neovascularisation within 48hours to weeks
- Causes subsequent reduction of IOP

Agents available:

- Ranibizumab – 0.5mg in 0.05ml
- Bevazicumab – 1.25mg in 0.05ml
- Aflibercept (eylea) – 2mg in 0.05ml
- Pegaptanib (macugen) – 0.3mg in 0.05ml

Most commonly used agents are ranibizumab and bevazicumab

Procedure:

- Under topical anesthesia, under sterile conditions
- Lid speculum applied
- Using a tuberculin syringe 0.05ml of the desired agent is injected
- Most commonly given in superotemporal quadrant
- Given at 3, 3.5,4 mm from limbus in cases of aphakic, pseudophakic and phakic eyes respectively

Complications:

- Subconjuctival hemorrhage
- Transient rise in intraocular pressure
- Intraocular inflammation
- Ocular haemorrhage
- Retinal detachment
- Endophthalitis
- Systemic events such as systemic hypertension, thromboembolic events etc.

SURGICAL MANAGEMENT:

- Indicated in patients with useful vision when IOP is not controlled with maximum medical therapy
- Pre op PRP, control of inflammation using steroids – essential

FILTERING SURGERIES:

- Higher failure rates due to increased inflammation, intraop haemorrhage and bleb failure due to fibrovascular proliferation
- Trabeculectomy with anti metabolites improves the outcome

- Glaucoma drainage devices are considered ideal

Anti metabolites:

Most commonly used are 5-Flurouracil(5FU) and Mitomycin C(MMC)

5 flurouracil:

- Pyrimidine analogue[7]
- Inhibits DNA synthesis
- Dose – 50mg/ml
- Soak in surgical sponges and placed on the sclera before creation of scleral flap
- Removed after 2 -4 minutes
- Excess washed away with Balanced Salt Solution
- Can also be used as subconjuctival injections

Mitomycin C:

- DNA crosslinker
- Inhibits fibroblast proliferation
- Dose – 0.2-0.4mg/ml
- Applied in a similar manner to 5 FU
- More potent than 5 FU
- Complications include scleral thinning and corneal toxicity

Steps of Trabeculectomy with Antimetabolites:

- Peribulbar block with lesser than usual amount given – to reduce the compromise of blood flow to optic nerve head[4]
- Lid speculum applied

- Bridle suture avoided as superior rectus hematoma can cause fibrovascular proliferation and bleb failure
- Inferior corneal traction suture is preferred
- A fornix based conjunctival flap is made
- Cotton sponges soaked in MMC or 5FU are placed on the scleral site for 2 to 4 minutes
- After that the sponges are removed and the area washed thoroughly with BSS[4]
- A partial thickness scleral flap is created and dissected till blue gray zone is reached
- A paracentesis done at 3 o clock position to avoid sudden decompression of globe
- At the scleral site, a 1.5 *2.5mm of trabecular tissue is excised with Kelly puch
- A peripheral iridectomy done taking care that iris doesnot plug the ostial site
- Scleral flap is sutuered with 10-0 nylon
- Conjuctival flap sutured with 10-0 nylon
- Saline injected through the sideport and bleb formed
- Injection Subconjuctival Dexamathosone 0.5cc given
- Topical antibiotic drops and sterile dressing applied

Complications of trabeculectomy:

- Hypotony with deep anterior chamber
    - Excessive inflammation
    - Leads to hypotony maculopathy
    - Autologous blood injection into bleb can be done to decrease filtration

- Hypotony with shallow anterior chamber:
    - Wound leak or bleb leak
    - Can be identified by Siedels test (Figure 19)
    - Prolonged shallow AC – endothelial decompensation
    - Pressure patch or Bandage contact lenses can be used
- Elevated IOP with deep anterior chamber:
    - Inadequate filtration
    - Ostium blocked with blood clot or iris
    - Argon laser may be used to clear the ostium
    - In case of blood clot, tPA may be used
- Elevated IOP with shallow anterior chamber:
    - Suprachoroidal haemorrhage
    - Pupillary block
    - Aqueous misdirection syndrome

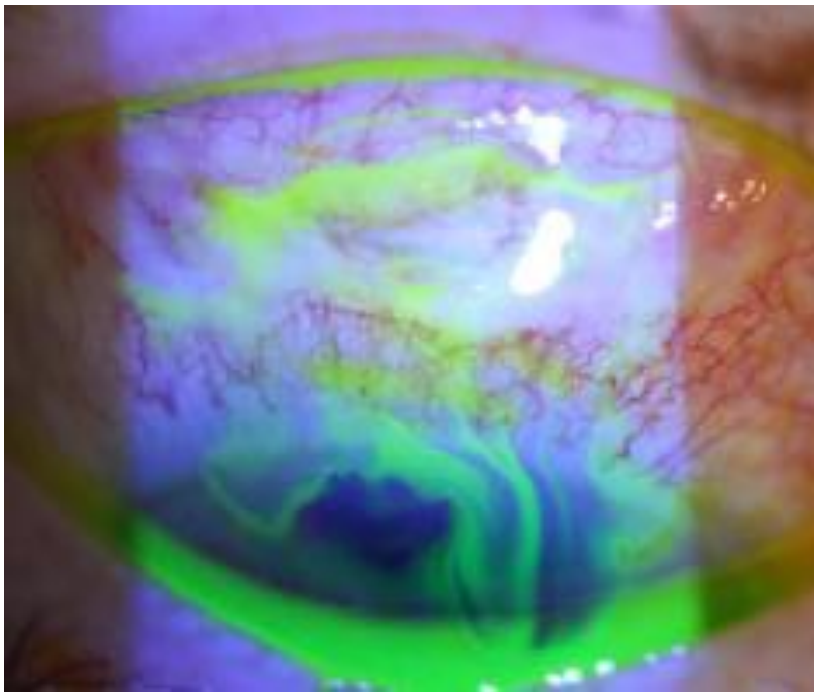


Figure 19: Diagram Showing Wound Leak with Siedel Sign Positive

Figure 20: Diagram Showing Ideal Bleb

Glaucoma Drainage Devices:

- Drainage devices with vitrectomy – better outcomes
- Can be classified into valved and non valved devices
- Non valved implants:
  - Molteno
  - Baerveldt (Figure 21)
- Valved implants:
  - Ahmed glaucoma valve (Figure 22)
  - Krupin
- Newer devices:
  - Istent
  - ExPRESS stent
  - Optimed glaucoma pressure regulator
  - Susanna glaucoma implant

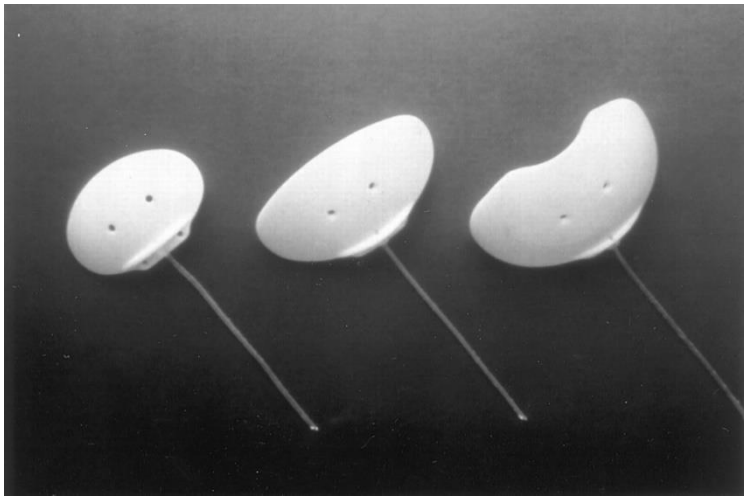


Figure 21: Diagram Showing Baerveldt Implant – Non Valved

Figure 22: Diagram Showing Ahmed Glaucoma Implant – Valved

Complications following Glaucoma drainage device implantation:

- Hypotony
- Valve malfunction
- Hyphaema
- Scleral perforation
- Tube related problems
- Tube erosion and endophthalmitis
- Migration or expulsion of the plate

- Corneal decompensation
- Overhanging blebs
- Strabismus

Cyclodestructive procedures:

- Done in eyes with no visual potential
- Done usually for pain relief
- Destroys the secretory epithelium of ciliary body
- Different modalities include – diathermy, cryotherapy, ultrasound and laser
- Cyclophotocoagulation - most commonly done
- Are of 2 types:
    - Trans scleral CPC
    - Endocyclophotocoagulation
- Done using semiconductor diode or Nd:Yag laser
- 6 spots per quadrant to a maximum of 18 spots are given
- No more than 3 quadrants – risk of anterior ischemia
- Placed till an audible pop is heard

Complications following cyclodestructive procedures:

- Transient pain
- Hyphaema
- Iridocyclitis
- Conjuctival burns
- Hypotony
- Pthisis bulbi

- Necrotising scleritis
- Malignant glaucoma
- Sympathetic opthalmia

RECENT ADVANCES:

- Photodynamic therapy with verteporfin – occlude iris new vessels without damaging the normal vasculature , but results not successful
- Systemic alpha interferon a polypeptide which inhibits proliferation and migration of endothelial cells has been tried
- Research studies are going on with regard to use of PEDF for NVG

AIM AND OBJECTIVES

AIM:

To analyse the demographic pattern, etiology, clinical presentation, treatment modalities and outcomes of neovascular glaucoma in a tertiary care centre

PRIMARY OBJECTIVES:

To determine the

- Etiology
- Stage of presentation
- Prognosis

In 100 cases of Neovascular Glaucoma

SECONDARY OBJECTIVES:

To study the

- demographic pattern,
- various treatment modalities and
- its complications in cases of neovascular glaucoma

MATERIALS AND METHODS

SUBJECT SELECTION:

100 cases of Neovascular Glaucoma who presented at Regional Institute of Ophthalmology and Government Ophthalmic Hospital, Egmore, Chennai were included in the study

INCLUSION CRITERIA:

Patients with Neovascular glaucoma ie;

- IOP > 21mmHg
- Neovascularization of Iris / Angle

EXCLUSION CRITERIA:

Patients with

- Primary open angle glaucoma
- Primary angle closure glaucoma
- Other forms of secondary glaucomas
- Age less than 18 years

Sample Size: 100 patients

Study centre: Glaucoma Services, RIO GOH, Egmore, Chennai

EXAMINATION METHODS:

Patients with neovascular glaucomas reporting to the glaucoma clinic RIOGOH wereregistered, consent obtained, evaluated and followed up during the study period. Visual acuity by Snellens chart, Anterior segment evaluation with slit lamp examination,

fundus examination by direct ophthalmoscopy and slit lamp biomicroscopy using 90D, Indirect ophthalmoscopy, IOP measurement by Goldmann applanation tonometer / rebound tonometer and gonioscopy using Goldmann single mirror examination were done for all patients. Routine investigations like Blood sugar, Urine analysis, blood pressure measurementswere done. Fundus Fluorescein Angiography to look for neovascularization and macular edema were done if there was a clear media. Cardiac evaluation and carotid Doppler were done wherever necessary to make the patient fit for for the planned management.

Final visual outcome and IOP were measured and recorded after the desired treatment given. Thorough examination of the patient during the follow up visits were done and complications of the treatment approaches were recorded.

RESULTS

1. AGE DISTRIBUTION:

Table 1: Age Distribution of Patients

Age in years	No. of Patients	Percentage
31-40	5	5
41-50	15	15
51-60	33	33
61-70	39	39
71-80	8	8

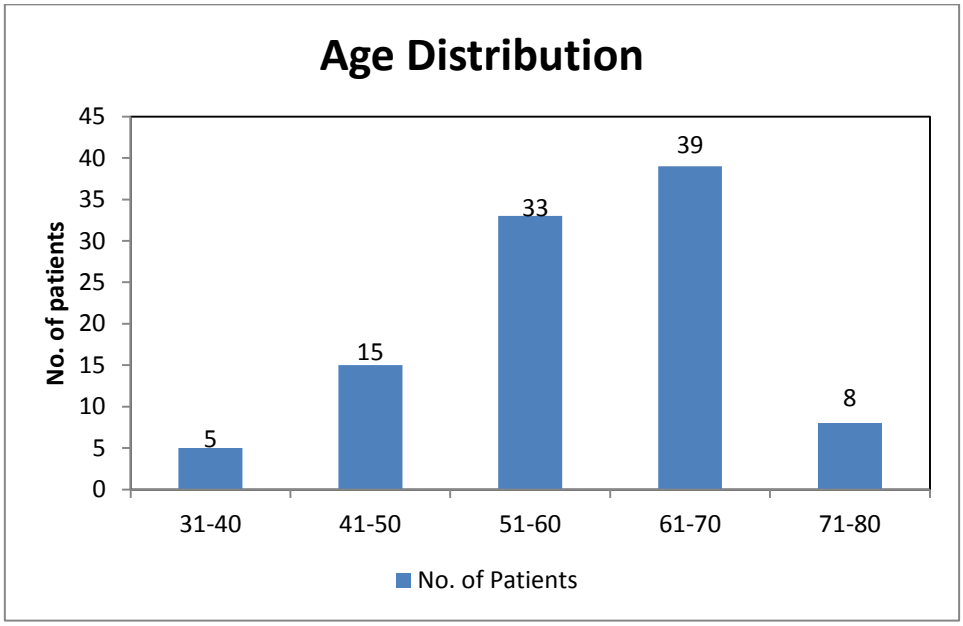


CHART 1: Age distribution of patients

In our study, out of 100 cases, 5% presented in 31 – 40 years age group, 15% presented in 41 – 50 years age group, 33% presented in 51 – 60 years age

group, 39% presented in 61 – 70 years age group and 8% in 71 – 80 years age group.Maximum number of cases (39%) were in 61 – 70 years group.

2. GENDER DISTRIBUTION:

Table 2: Gender Distribution

Gender	No. of patients	Percentage
Male	60	60
Female	40	40

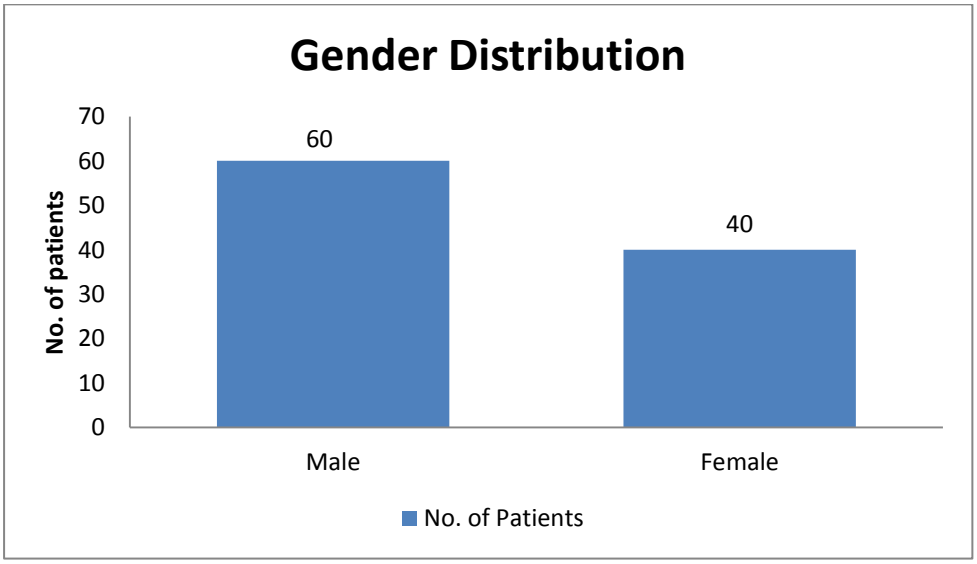


Chart 2: Gender Distribution

In this study, among 100 cases, 60% were males and 40% were females

3. EYE INVOLVED:

Table 3: Eye Involved

Eye Involved	No. of Patients	Percentage
RE	63	63
LE	37	37

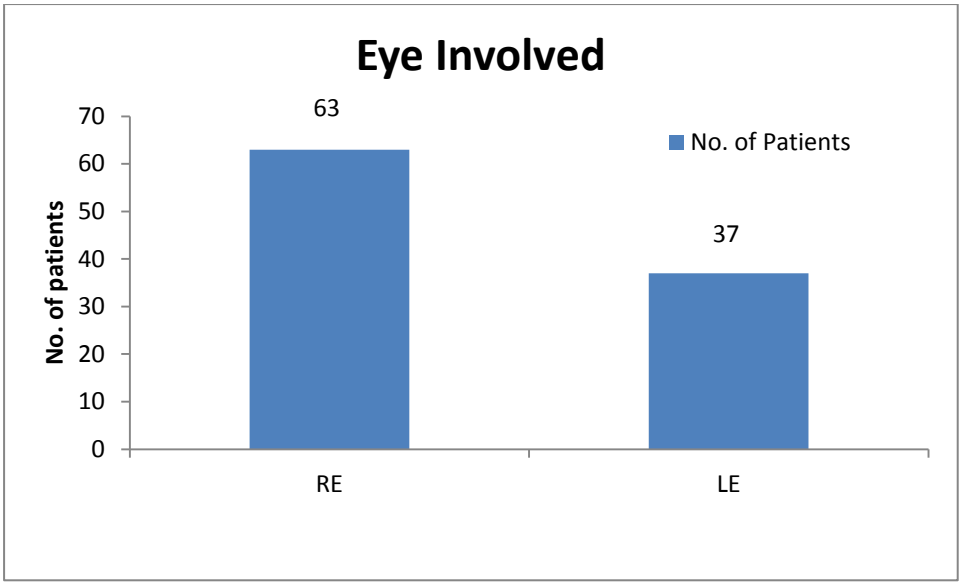


Chart 3: Eye Involved

In this study, 63% had right eye involvement and 37% had left eye involvement

4. VISION AT THE TIME OF PRESENTATION

Table 4: Visual Acuity at the Time of Presentation

Vision at Presentation	No. of Patients	Percentage
NOPL	8	8
PL+	21	21
HM	16	16
CFCF	5	5
1/60-3/60	28	28
4/60-6/60	14	14
>6/60	8	8

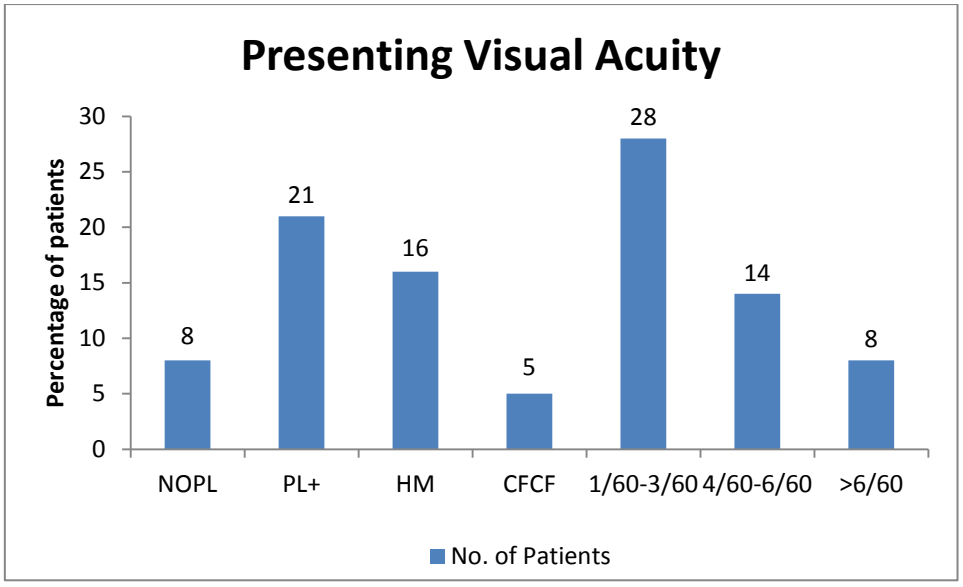


Chart 4: Visual Acuity at the Time of Presentation

In this study, visual acuity at the time of presentation was NO PL in 8% of cases, PL + in 21%, HM in 16%, CFCF in 5%, between 1/60-3/60 in 28%, between 4/60-6/60 in 14% and >6/60 in 8% of cases.

Maximum number of cases (28%) had visual acuity between 1/60 – 3/60

5. INTRA OCULAR PRESSUREAT PRESENTATION

Table 5: IOPat the Time of Presentation

IOP at presentation (mmHg)	No. of Patients	Percentage
21-30	19	19
31-40	35	35
41-50	35	35
51-60	11	11

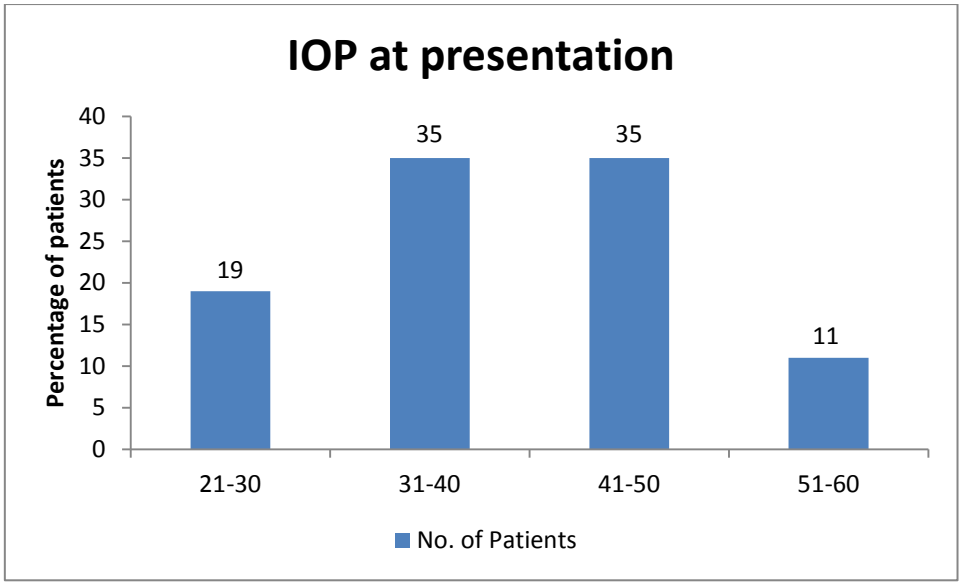


Chart 5: IOPat the Time of Presentation

In this study, 19% had IOP between 21 to 30mmHg, 35% had IOP between 31-40mmHg, 35% had IOP between 41 to 50mmHg, 11% had IOP between 51 to 60mmHg.

Maximum number of cases presented with IOP between 31 to 50mmHG

6. STAGE OF PRESENTATION

Table 6: Stage of Presentation

Stage of presentation	No. of patients	Percentage
OAG	40	40
ACG	52	52
AG	8	8

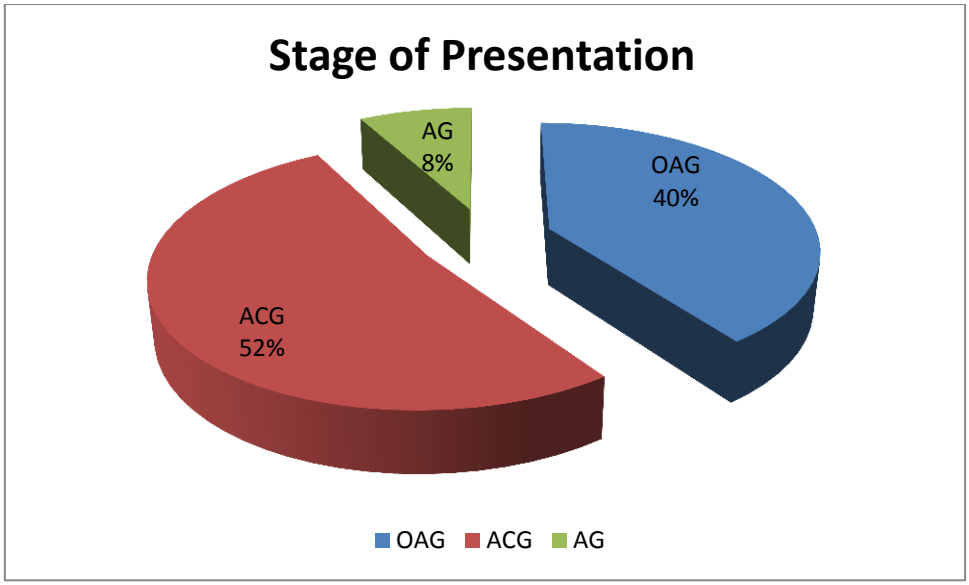


Chart 6: Stage of Presentation

In this study, among 100 cases, 40% presented in Open angle Glaucoma stage, 52% presented in Angle closure Glaucoma stage and 8% presented in Absolute Glaucoma stage

Maximum number of patients (52%) presented in Angle closure Glaucoma stage.

7. ETIOLOGY

Table 7: Etiology of Neovascular Glaucoma

Etiology	No. of Patients	Percentage
PDR	37	37
CRVO	22	22
BRVO	6	6
CRAO	2	2
UVEITIS	17	17

POAG	8	8
PACG	4	4
PXFG	2	2
UGH	1	1
POSTRD	1	1

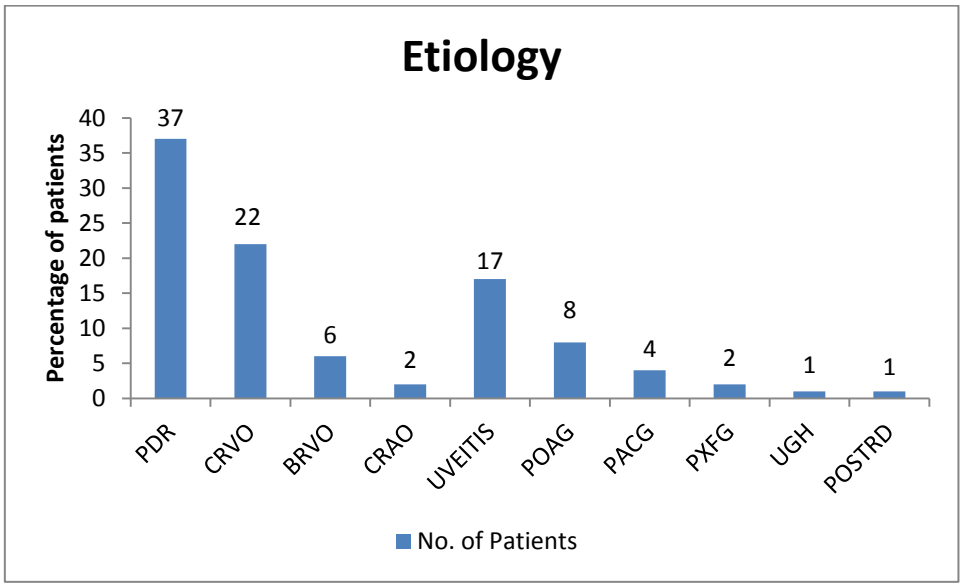


Chart 7: Etiology of Neovascular Glaucoma

In this study, Diabetic Retinopathy was found to be the etiology for 37% of cases, Vascular occlusion was found to contribute to 30% (including CRVO – 22%, BRVO – 6, CRAO – 2%), chronic uveitis contributed to 17% of cases, Glaucoma was found to be the etiology in 14% cases (including POAG – 8%, PACG – 4%, PXF glaucoma – 2%). Other causes like Uveitis Glaucoma Hyphaema syndrome and post RD surgery were found in 2 cases,

Maximum number of cases had Diabetic retinopathy as etiology – 37%, followed closely by Vascular occlusion – 30%

8. VARIOUS TREATMENT MODALITIES

Table 8: Various Treatment Modalities

Treatment	No. of Patients	Percentage
PRP	26	26
TRAB+AM	36	36
GDD	4	4
ANTI VEGF	6	6
MEDICAL	23	23
IOLR+TRAB+AM	1	1
CYCLO CRYO	4	4

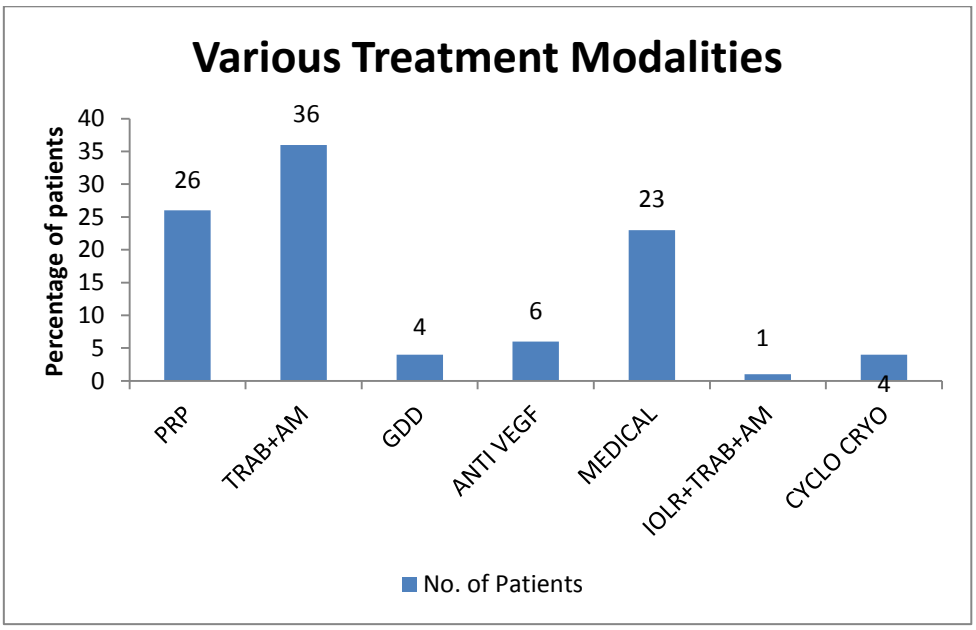


Chart 8: Various Treatment Modalities

In this study, out of the 100 neovascular glaucomas presented to us, 36% of patients required Trabeculectomy with antimetabolites, 4% cases underwent

Glaucoma Drainage Device (Ahmed Glaucoma Valve)implantation, 1 case of UGH underwent ACIOL removal with Trabeculectomy with antimetabolites, 26% of cases underwent panretinal photocoagulation, 23% patients were on medical treatment only, 6% of patients were given intravitreal anti VEGF and 4% of patients had no useful visual potential and underwent Cyclocryotherapy.

Most commonly employed intervention was Trabeculectomy with antimetabolites (36%) indicating the need for surgical intervention in Neovascular Glaucoma

9. TREATMENT MODALITIES IN OAG GROUP

Table 9: Treatment Modalities in OAG Group

Treatment	No. of Patients	Percentage
PRP	17	42.50%
TRAB+AM	7	17.50%
MEDICAL	9	22.50%
ANTI VEGF	6	15%
IOLR+TRAB+AM	1	2.50%

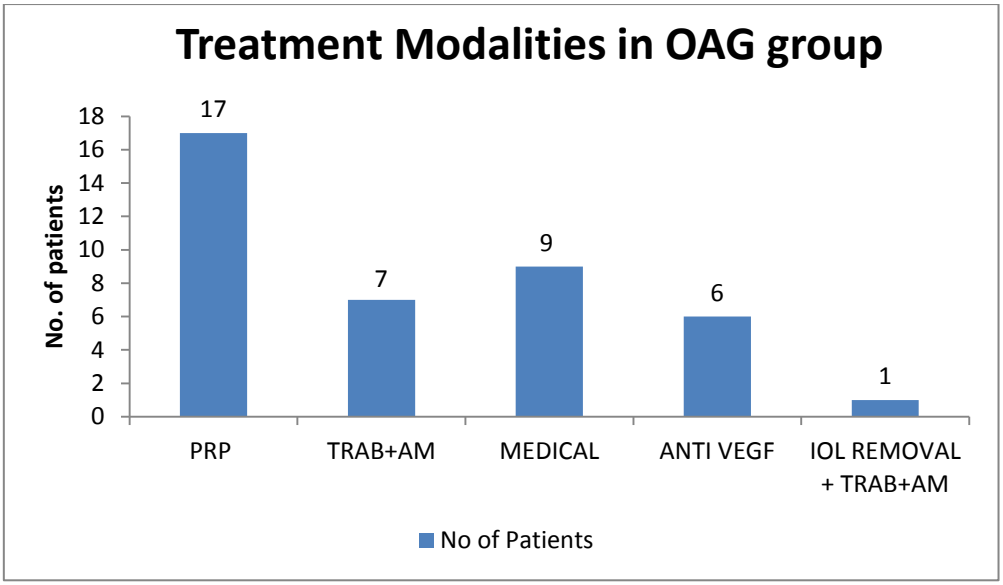


Chart 9: Treatment Modalities in OAG Group

In this study, out of the 40 patients who presented in Open angle Glaucoma stage, 42.50% patients underwent pannretinal photocoagulation, 22.50% were maintained on medical treatment alone, 17.50% required trabeculectomy with antimetabolites, 15% were given Intravitreal anti VEGF injections, and 2.50% needed IOL removal with Trabeculectomy with antimetabolites.Thus, patients presenting in Open angle stage were managed mostly with PRP and medical treatment and only 17.50% required filtering surgery.

10. TREATMENT MODALITIES IN ACG GROUP

TABLE 10: TREATMENT MODALITIES IN ACG GROUP

Treatment	No. of Patients	Percentage
PRP	9	17.30%
TRAB+AM	29	55.77%
GDD	4	7.70%
MEDICAL	10	19.23%

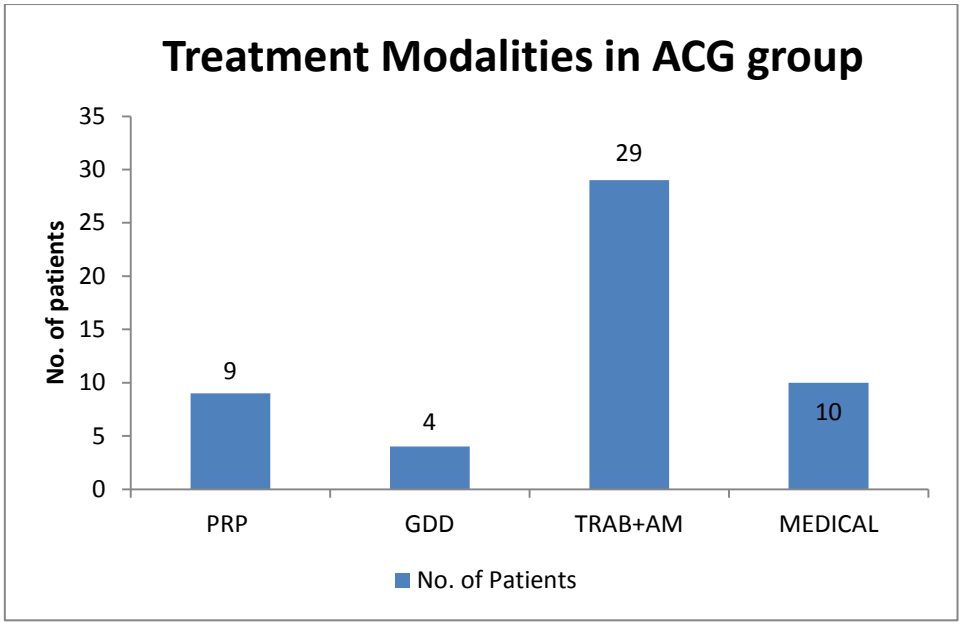


Chart 10: Treatment Modalities in ACG Group

In this study, out of 52 patients who presented in angle closure stage, 55.77% required Trabeculectomy with antimetabolites, 7.70% were taken up for Ahmed valve implantation, 19.23% were on medical treatment only and 17.30% underwent PRP

Thus, in patients presenting in angle closure stage surgical interventions were required in maximum number of cases(63.47%) rather than medical or laser treatment.

11. TREATMENT MODALITIES IN AG GROUP

Table 11: Treatment Modalities in AG Group

Treatment	No. of Patients	Percentage
MEDICAL	4	50%
CYCLOCRYO	4	50%

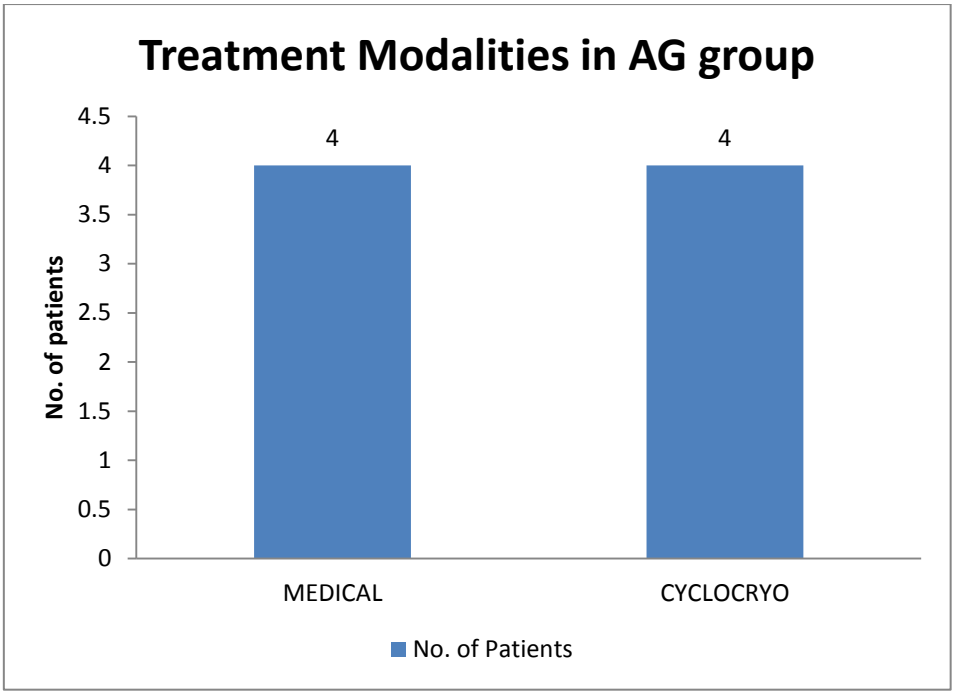


Chart 11: Treatment Modalities in AG Group

In this study, out of 8 cases who presented in absolute glaucoma stage, 50% underwent cyclocryo and 50% were maintained on medical treatment alone

12. VISUAL OUTCOME IN OAG GROUP

Table 12: Visual Outcome in OAG Group

Vision	No. of Patients	Percentage
<6/60	12	30%
6/60	8	20%
>6/60	20	50%

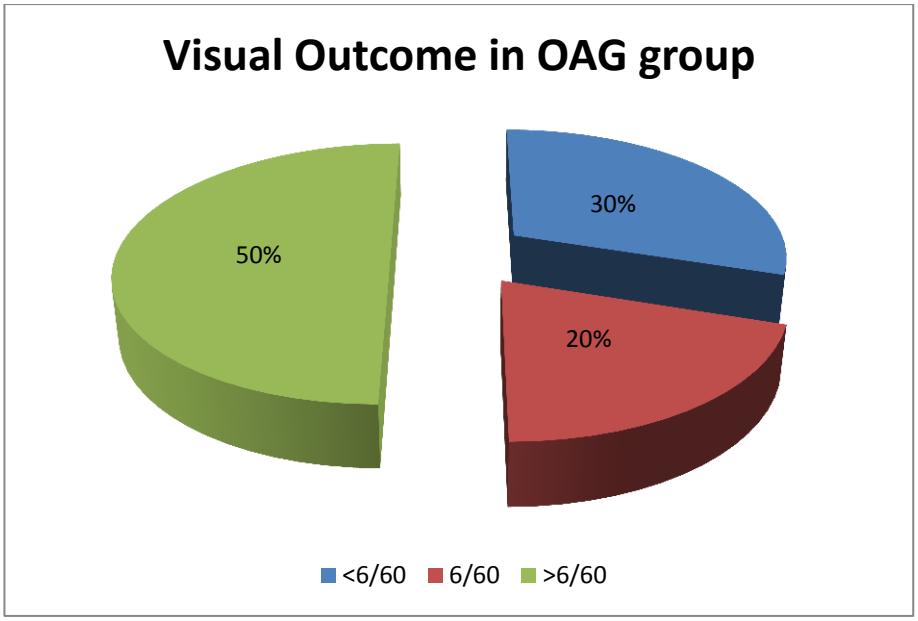


Chart 12: Visual Outcome in OAG Group

In this study out of 40 patients presenting in open angle stage, 50 % had good visual outcome of >6/60, 20% had a vision of 6/60 and 30% had vision <6/60 Thus, around 50% patients improved to have a vision>6/60

13. VISUAL OUTCOME IN ACG GROUP

Table 13: Visual Outcome in ACG Group

Vision	No. of Patients	Percentage
<6/60	29	55.77%
6/60	9	17.31%
>6/60	14	26.92%

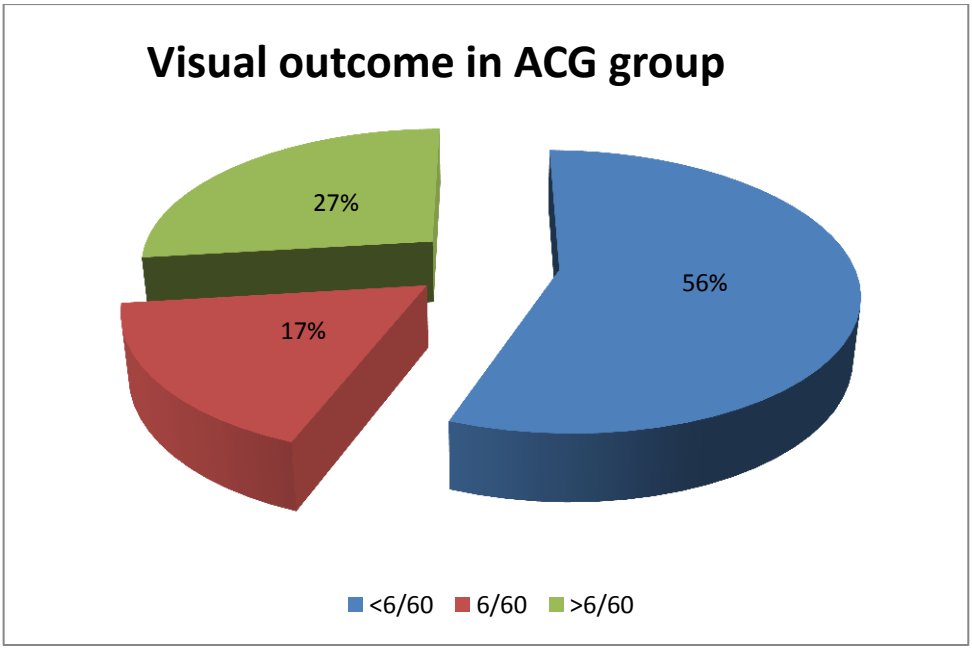


Chart 13: Visual Outcome in ACG Group

In this study, out of 52 patients who presented in angle closure stage, only 26.92% had better vision of >6/60, 17.31% had vision of 6/60 and 55.77% had poor vision of <6/60

Thus, patients presenting in angle closure stage, around 56% had poor final visual outcome

14. IOP CONTROL IN OAG GROUP

Table 14: IOP Control in OAG Group

IOP	No. of Patients	Percentage
<20	3	7.5%
20-30	32	80%
>30	5	12.5%

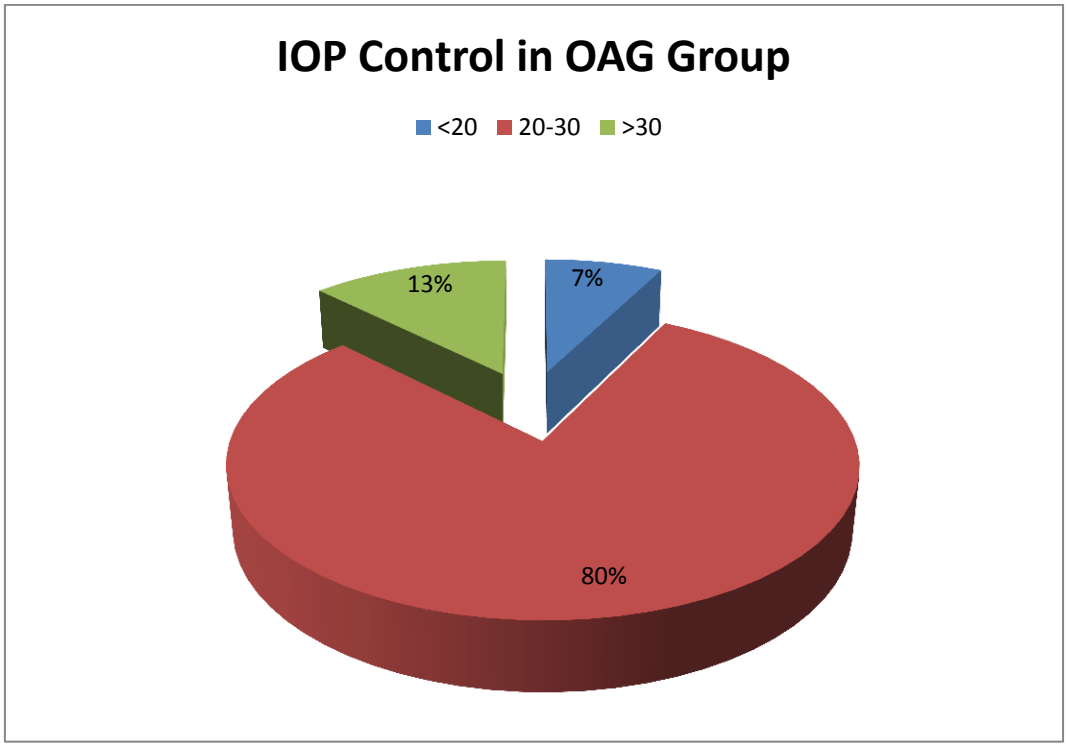


Chart 14: IOP Control in OAG Group

In this study, 7.5% had very good IOP control <20mmHg, 80% had fair IOP control between 20 – 30mmHg, and 12.5% had poor control with IOP >30mmHg

Thus, among Open angle patients, 87.5% had fair IOP control of <30mmHg

15. IOP CONTROL IN ACG GROUP

Table 15: IOP Control in ACG Group

IOP	No. of Patients	Percentage
<20	2	4%
20-30	24	46%
>30	26	50%

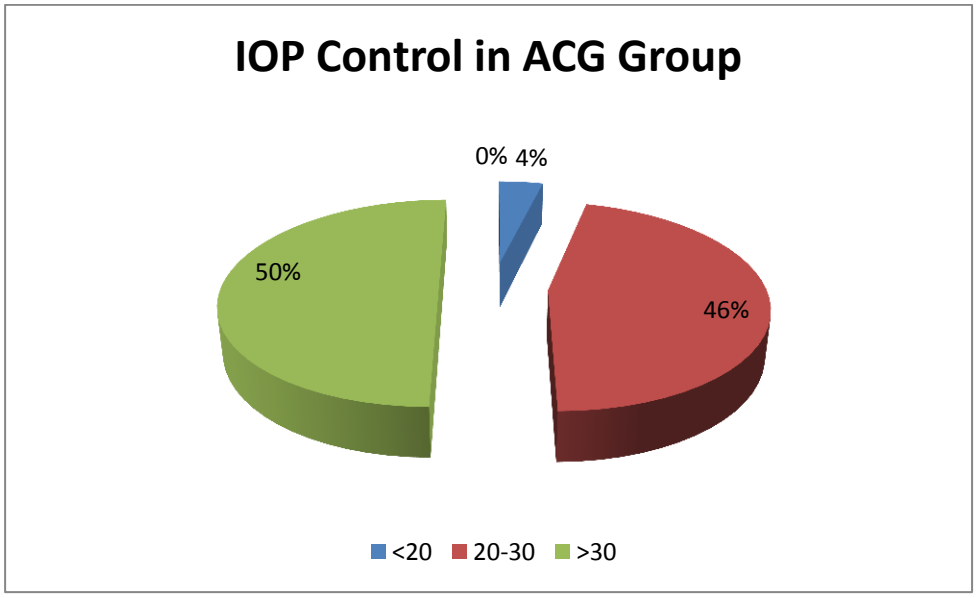


Chart 15: IOP Control in ACG Group

In this study, among patients presenting in angle closure stage 4% had good IOP control of <20mmHg, 46% had fair control with IOP between 20 to 30mmHg, and 50% had poor control with IOP>30mmHg

COMPLICATIONS OF VARIOUS TREATMENT MODALITIES

16. COMPLICATIONS OF PANRETINAL PHOTOCOAGULATION

Table 16: Complications of Panretinal Photocoagulation

Complications	No. of Patients	Percentage
None	3	12%
Iritis	23	88%
Transient Rise in IOP	10	38%
Vitreous Haemorrhage	1	4%

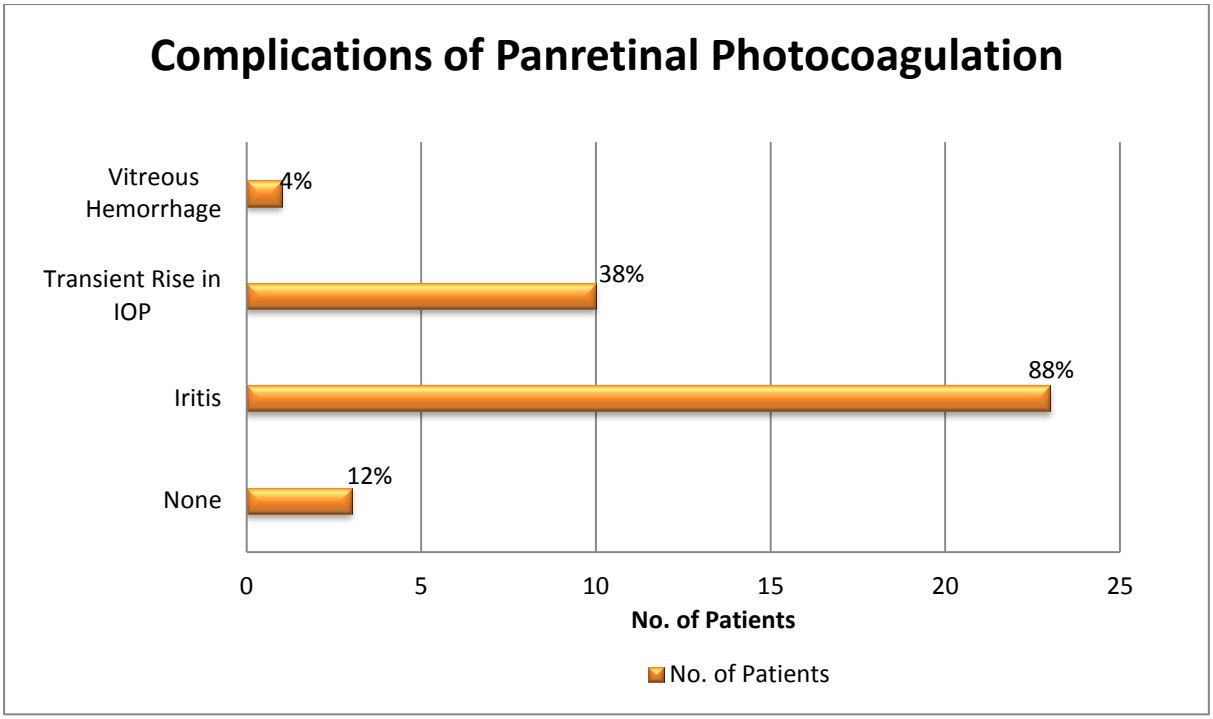


Chart 16: Complications of Panretinal Photocoagulation

In this study, among 26 patients who underwent Panretinal Photocoagulation, 12% had no complications, 88% had Iritis, 38% had transient increase in IOP and 4% had vitreous haemorrhage

Thus, most common complication following PRP was found to be Iritis (88%)

17. COMPLICATIONS OF TRABECULECTOMY + ANTIMETABOLITES:

Table 17: Complications of Trabeculectomy + Antimetabolites

Complications	No. of Patients	Percentage
Iritis	33	92%
Shallow AC	18	50%
Hyphaema	12	33%
Choroidal detachment	8	22%

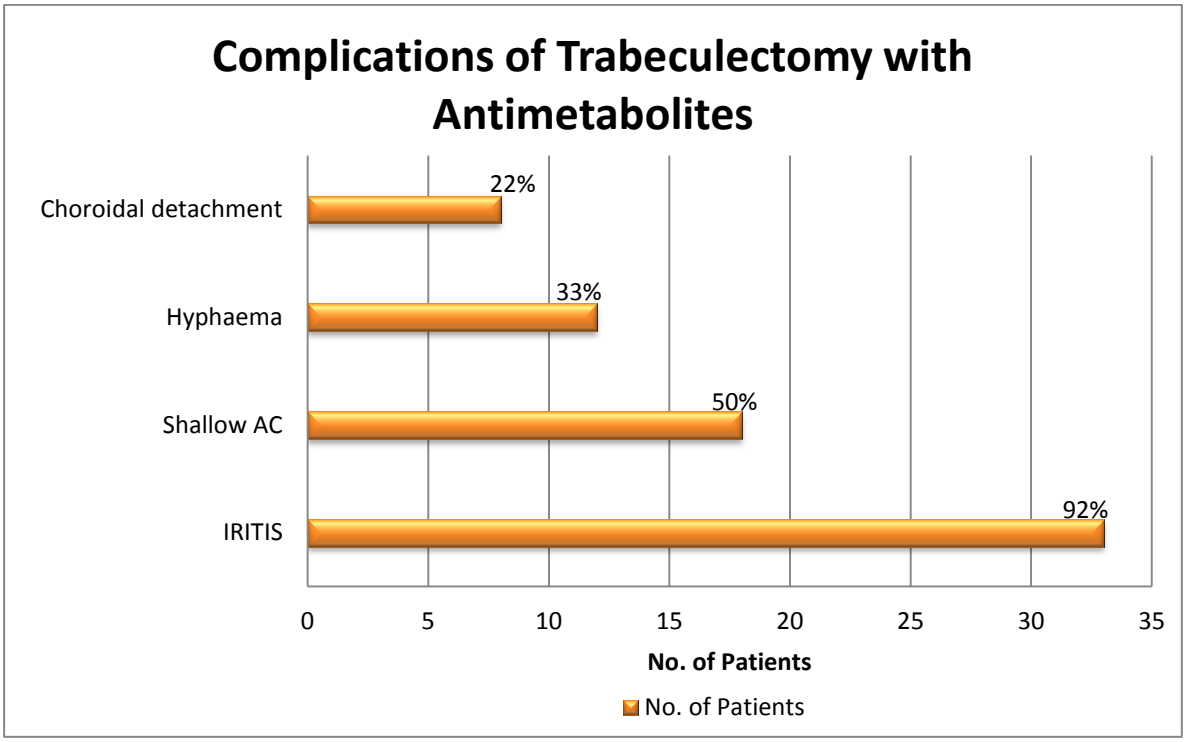


Chart 17: Complications of Trabeculectomy +Antimetabolites

In this study, among 36 cases who underwent Trabeculectomy with anti metabolites, 92% had Iritis, 50% had shallow anterior chamber, 33% had hyphaemaand 22% had choroidal detachment. Thus, most common complications following filtering surgery was found to be Iritis and shallow anterior chamber

18. COMPLICATIONS OF GLAUCOMA DRAINAGE DEVICE

Table 18: Complications of GDD

Complications	No. of Patients	Percentage
Iritis	3	75%
Shallow AC	1	25%
Hyphaema	1	25%

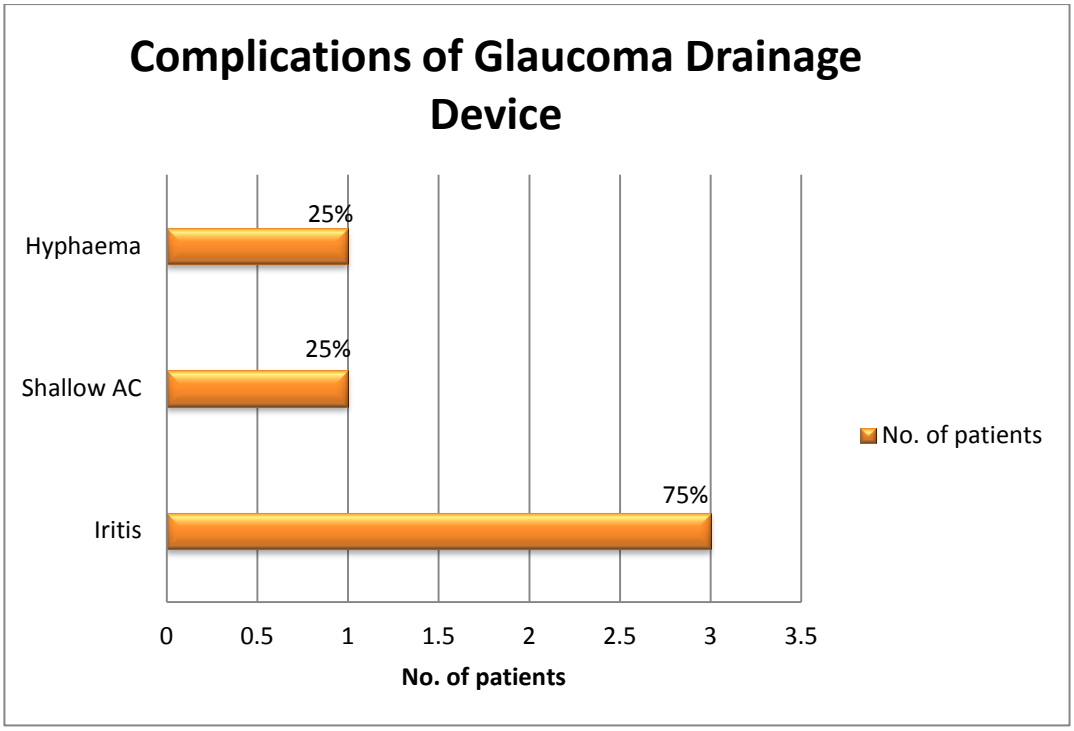


Chart 18: Complications of GDD

In this study, out of 4 patients who underwent Ahmed Glaucoma Valve implantation, 75% had iritis, 25% had shallow anterior chamber and 25% had hyphaema.

19. COMPLICATIONS OF ANTI VEGF

Table 19: Complications of Anti VEGF

Complications	No. of Patients	Percentage
None	3	50%
Transient Rise in IOP	3	50%

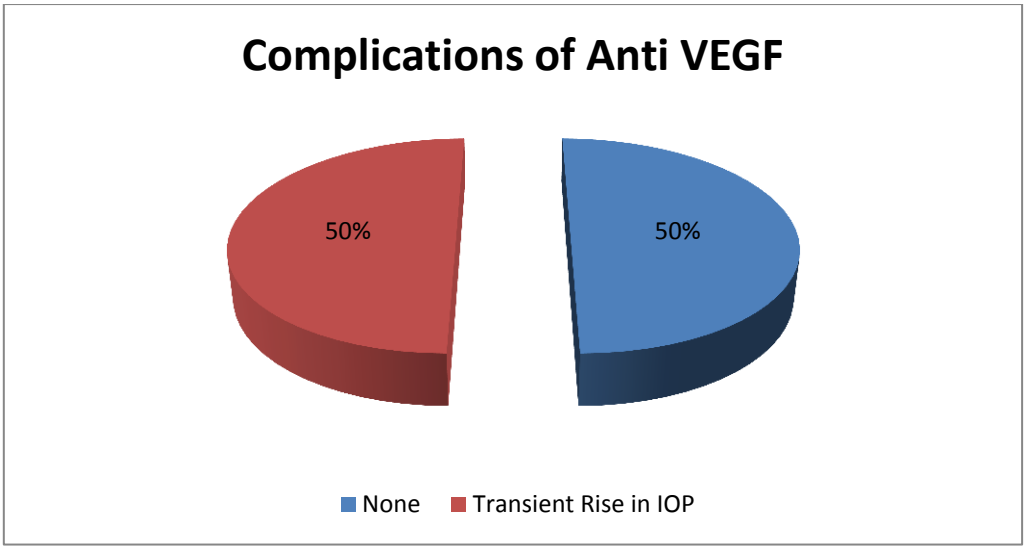


Chart 19: Complications of Anti VEGF

In this study among 6 patients who underwent intra vitreal anti VEGF injections, 50 % had no complications and 50% had transient increase in IOP which came back to normal after medical treatment.

20. COMPLICATIONS OF MEDICAL TREATMENT:

Table 20: Complications of Medical Treatment

Complications	No. of Patients	Percentage
None	17	73.90%
Conjuctival Hyperemia	6	26.08%
Persistent Pain	6	26.08%

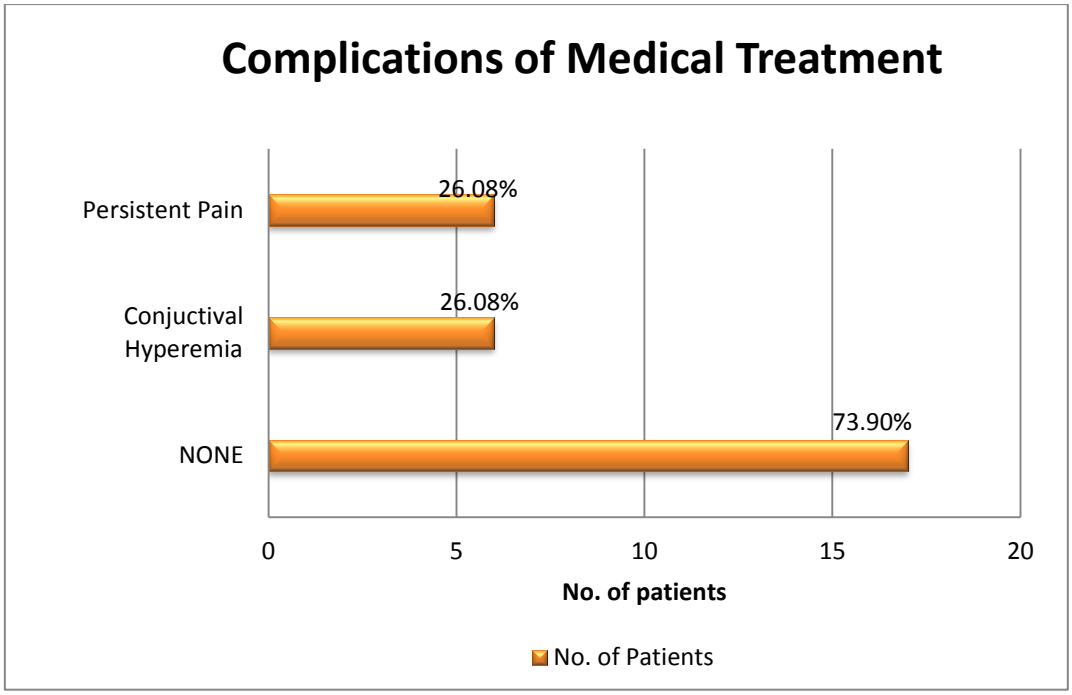


Chart 20: Complications of Medical Treatment

In this study, among 23 patients who were on medical treatment alone, 73.90% had no significant complications, 26.08% had conjunctival hyperemia and 26.08% had persistent pain

21. FINAL VISUAL OUTCOME :

Table 21: Final Visual Outcome

Final Visual acuity	No. of Patients	Percentage
≤6/60	66	66
>6/60	34	34

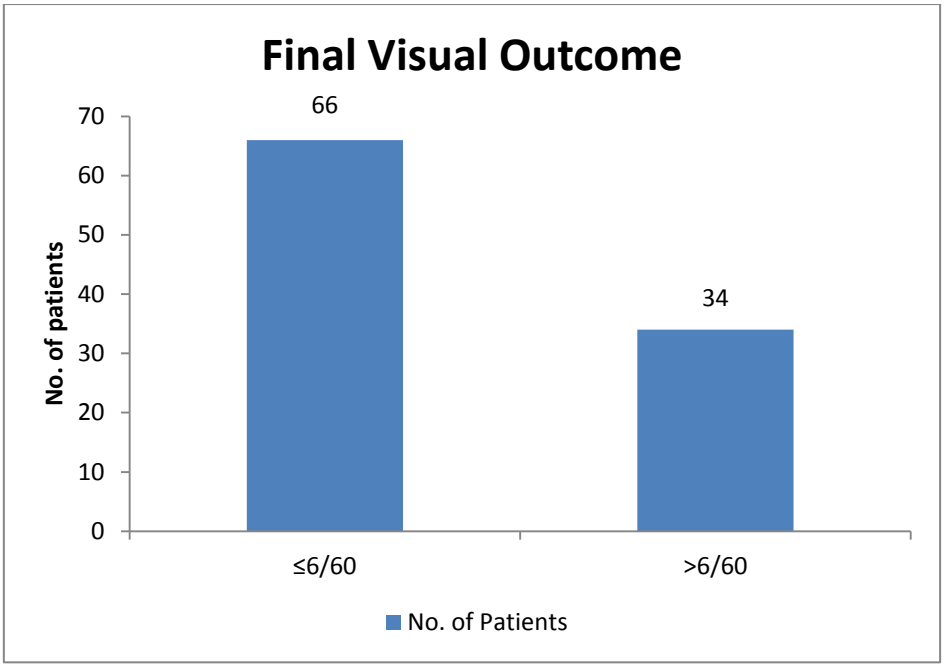


Chart 21: Final Visual Outcome

In this study, after undergoing all the various modalities of treatment, only 34% had a better vision of >6/60 and 66% had poor visual outcome of ≤6/60

22. FINAL IOP CONTROL

Table 22: Final Intraocular Pressure

Final IOP	No. of Patients	Percentage
Hypotony	4	4
≤20	5	5
21-30	61	61
>30	30	30

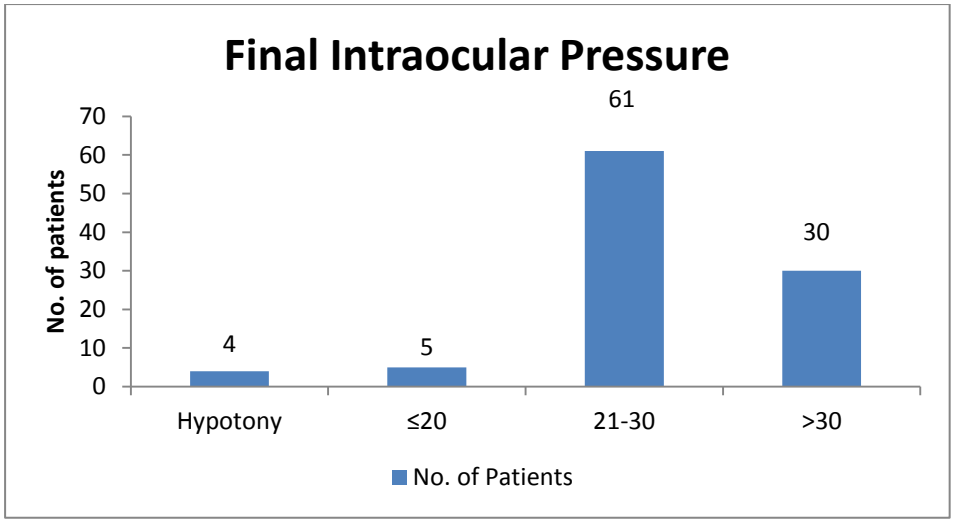


Chart 22: Final Intraocular Pressure

In this study, among 100 cases, after all the treatment modalities, 4% developed hypotony following cyclocryo, 5% had a good control of IOP of <20mmHg, 61% had a fair control with IOP between 20-30mmHg, and 30% continued to have persistent high IOP of >30mmHg

23. COMPARISON OF VISUAL ACUITY AT PRESENTATION AND FINAL VISUAL OUTCOME

Table 23: Comparison of Visual Acuity at Presentation and Final Visual Outcome

			VN		
			FINAL	PRESEN	Total
Category	≤6/60	Count	66	92	158
		% within VN	66.0%	92.0%	79.0%
	>6/60	Count	34	8	42
		% within VN	34.0%	8.0%	21.0%
Total		Count	100	100	200
		% within VN	100.0%	100.0%	100.0%

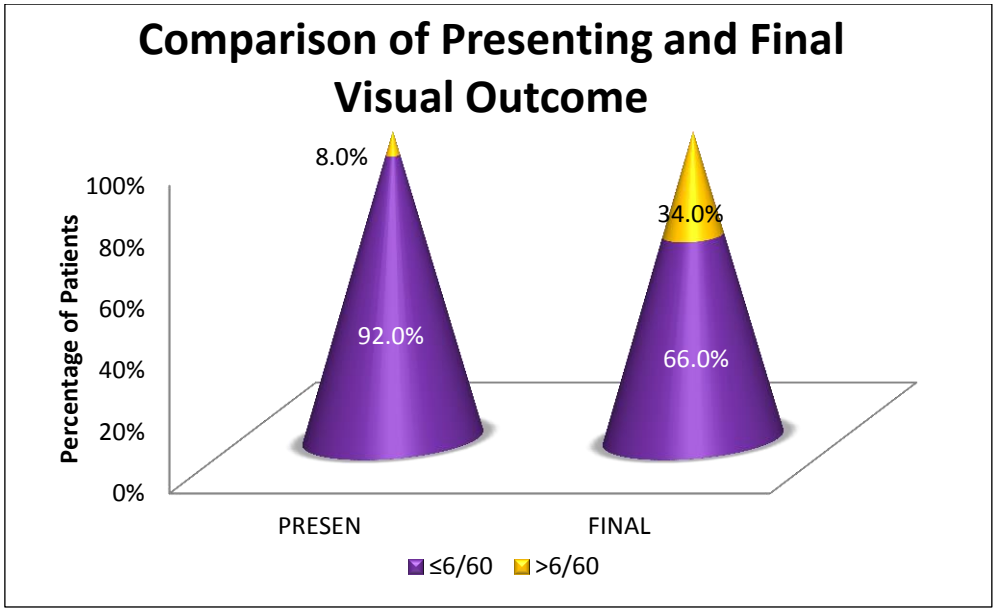


Chart 23: Comparison of Visual Acuity at Presentation and Final Visual Outcome

In this study, out of 92 patients, who presented to us with a poor vision of ≤6/60, only 26 improved to have better vision of > 6/60 after employing the above said modalities of treatment, while 66 patients persisted to have poor vision of ≤6/60. The statistical comparison was done using Chi-square test and the was found to be statistically significant with p=0.0005 (p<0.05 is significant)

24. COMPARISON OF IOP AT PRESENTATION AND FINAL IOP

Table 24: Comparison of IOPat Presentation and Final IOP

		IOP		Total
		FINAL	PRESENT	
Category	≤ 30	70	19	89
	>30	30	81	111
Total		100	100	200

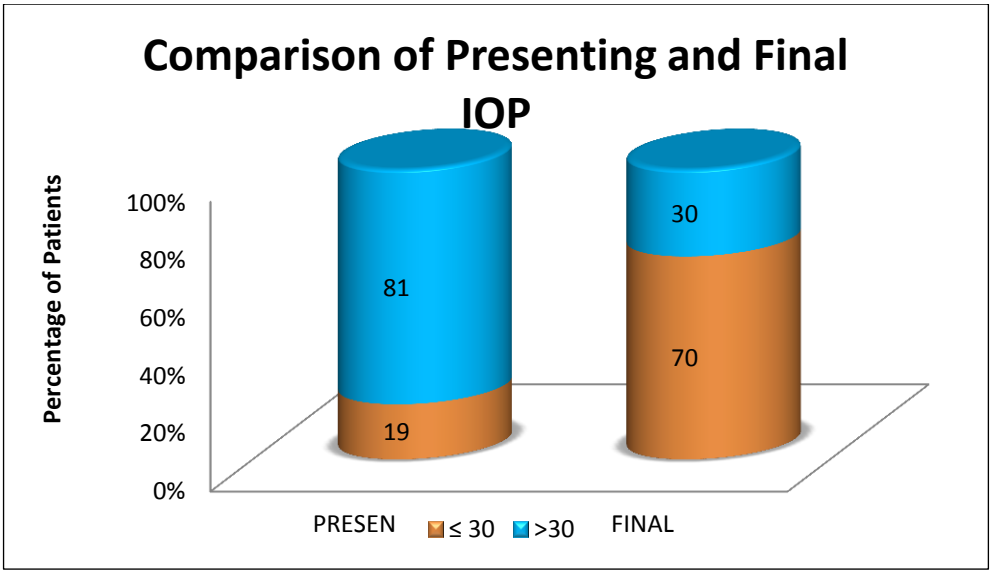


Chart 24: Comparison of IOPat Presentation and Final IOP

In this study, among the 81 patients who presented with a very high IOP of >30mmHg, only 51 patients attained a good control with a final IOP of ≤30mmHg, while 30 patients had a persistent elevation of more than 30mmHg.The statistical comparison was done using Chi-square test and it was found to be statistically significant with p=0.0005 (p<0.05 is significant)

25. COMPARISON OF VISUAL OUTCOMES IN OAG AND ACG GROUP

Table 25: Comparison of Visual Outcomes in OAGand ACG Group

			Stage of Presentation		Total
			ACG	OAG	
Results	VN≤6/60	Count	38	20	58
		% within Presentation	73.1%	50.0%	63.0%
	VN>6/60	Count	14	20	34
		% within Presentation	26.9%	50.0%	37.0%
Total		Count	52	40	92
		% within Presentation	100.0%	100.0%	100.0%

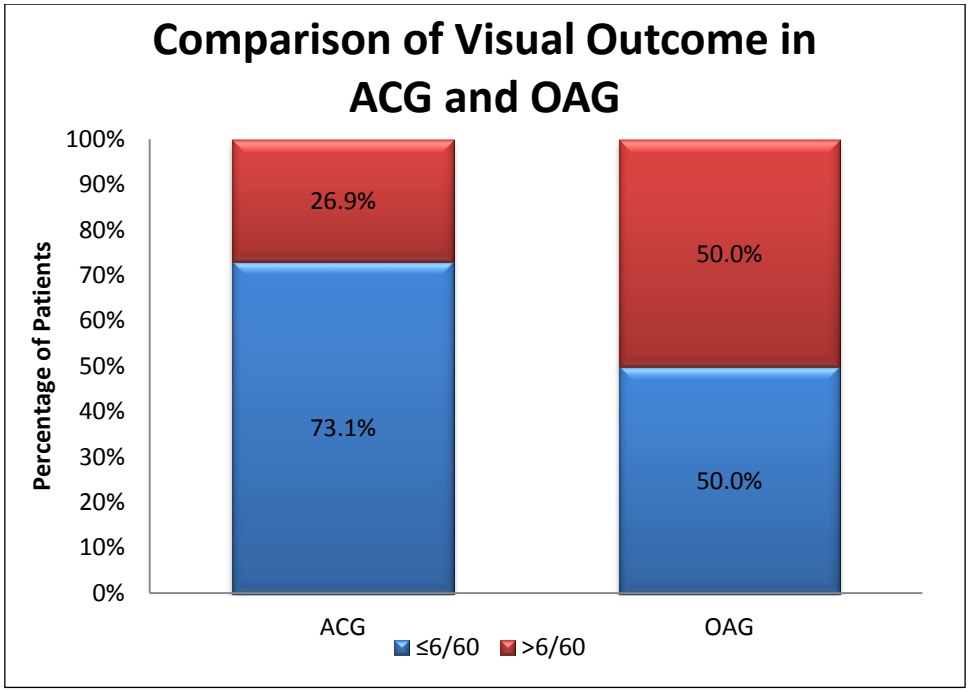


Chart 25: Comparison of Visual Outcomes in OAGand ACG Group

In this study, among 40 patients who presented in open angle stage, 50% had good visual outcome with vision >6/60, while 50% had poor vision. Among 52 patients who presented in angle closure stage, only 26.9% had better vision of more than 6/60, while 73.1% had persistent poor vision of ≤6/60.

The statistical comparison was done using Chi-square test and the was found to be statistically significant with p=0.023 (p<0.05 is significant)

26. COMPARISON OF IOP CONTROL IN OAG AND ACG GROUP

Table 26: Comparison of IOP Control in OAGand ACG Group

			Presentation		Total
			ACG	OAG	
Results	IOP≤30	Count	26	35	61
		% within Presentation	51.9%	87.5%	67.4%
	IOP>30	Count	26	5	31

		% within Presentation	48.1%	12.5%	32.6%
Total		Count	52	40	92
		% within Presentation	100.0%	100.0%	100.0%

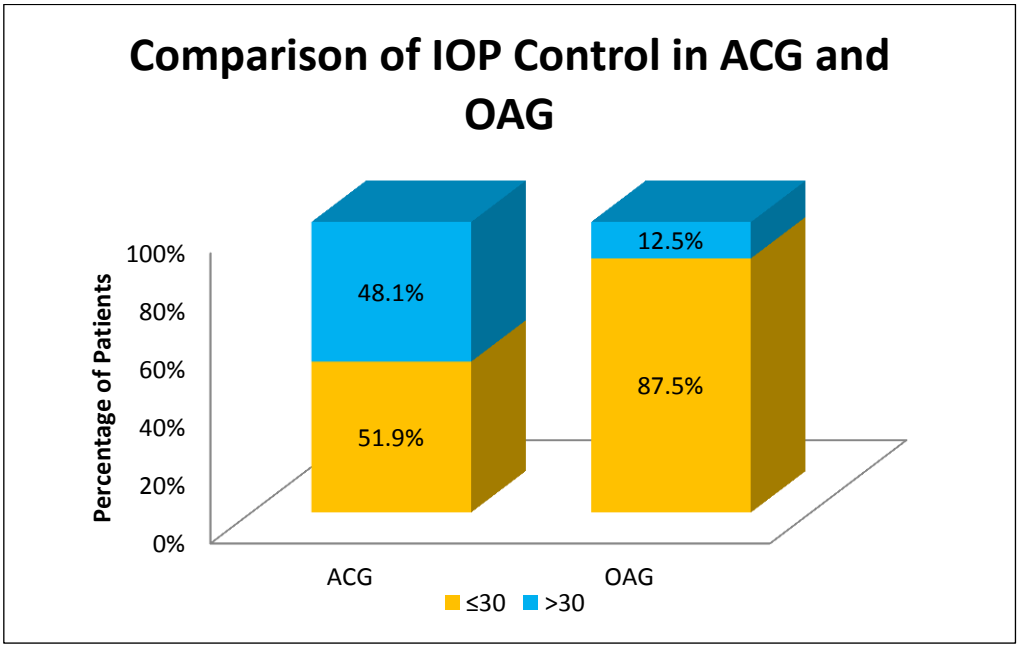


Chart 26: Comparison of IOP Control in OAGand ACG Group

In this study, among 40 patients who presented in open angle stage, 87.5% attained a fair control of IOP with final IOP ≤ 30mmHg. Among 52 patients who presented in angle closure stage, only 50% attined fair control of IOP≤30mmHg while 50% had persistent high IOP of more than 30mmHg. The statistical comparison was done using Chi-square test and the was found to be statistically significant with p=0.0005 (p<0.05 is significant)

DISCUSSION

1. **Age:**In our study, maximum number of patients(39%) presented in the age group between 61-70 years. In a study by Gustavo B. Rodrigues et al, it has been shown that prevelance was maximum in > 60 years age group[17]
2. **Gender:** In our study, there was a male preponderance (M:F=60:40). Previous studies have also shown a slight male preponderance but it was not statistically significant.[17]
3. **Eye Involved:** In our study, around 63% had right eye involvement while 37% had left eye involvement.

   Previous studies have shown no significant preponderance regarding the eye involved.[17]
4. **Visual acuity at the time of presentation:** In our study, 8% had NOPL, 42% had <1/60, 28% had vision between 1/60-3/60, 14% had vision between 3/60-6/60 and only 8% presented with vision>6/60.

   Previous studies have also indicated the disease to have severe visual loss with maximum percentage of patients having vision of only perception of light at presentation.[18]
5. **Intraocular pressure at the time of presentation:** In our study, maximum number 70% had IOP range of 30-50mmHg suggesting the severity of the disease.

   Previous studies have also shown a high IOP at the time of presentation.[17][18]

6. **Stage of presentation**: In our study, 40% presented in Stage 3 (open angle), 52% presented in Stage 4(angle closure) and 8% in Stage 5(absolute stage) of Neovascular glaucoma.

   Previous studies have also shown the advanced stage of presentation of these patients in a tertiary hospital.[17]

7. **Etiology:** In our study, the most common etiology was found to be Diabetic retinopathy(37%) followed closely by vascular occlusion(30%) suggesting retinal ischemia to be the most common cause of neovascular glaucoma.

   Previous studies have also shown Diabetic retinopathy and vascular ischemic events to be the major causative factors in Neovascular glaucoma[15][19]

8. **Various treatment modalities:** In our study, around 41% required surgical procedures (filtering surgery with antimetabolites and Ahmed valve implantation) indicating the need for surgical intervention in maximum number of cases.

   Previous studies have also shown that it is refractory to medical treatment alone and ultimately necessitates surgery[17]

9. **Treatment approaches according to stage of presentation:** In our study, the most common treatment modality employed in open angle stage group was panretinal photocoagulation(42.50%), whereas it was surgical intervention(63.47%) in angle closure group. This shows that surgical intervention was needed when patients presented in advanced stage rather than medical and laser treatment which were found to be useful in open angle patients as they present early in the course of disease.

Previous studies have emphasised the need for surgeries in advanced presentation groups.[17]

10. **Visual outcomes in open angle and angle closure group:** In our study, the open angle group had a final better visual outcome of more than 6/60 in 50% of cases rather than the meagre 26.9% cases who had better vision of more than 6/60 in angle closure group. It was also statistically significant with $p<0.05\%$. This suggests visual improvement was better in open angle than angle closure group. This is due to the fact, the open angle group have presented early in the course of their disease.

11. **Intraocular Pressure control in open angle and angle closure group:**

In our study, open angle patients had a fair Intraocualr pressure control of ≤ 30mmHg in 87.5% of patients, whereas angle closure group attained Intraocular pressure≤ 30mmHg in only 50% of patients which clearly shows the failure to attain IOP control in angle closure stage.It was also statistically significant with $p<0.05\%$.

This is again due to the fact that open angle glaucoma stage is an early stage and thus patients are better managed when presented early. Various studies have clearly indicated angle closure to have a marked impact on final Intaocular pressure control. In a study by Satoko Nakano et al, angle closure has been identified to have the maximum impact on Intraocular pressure control.[16]

12. **Complications of various treatment modalities employed:**

- **Complications of PRP:** In patients who were given PRP, 88% had iritis, 38% had transient increase in IOP and 4% developed vitreous haemorrhage.
- **Complications of Trabeculectomy with antimetabolites**: In 36 patients who underwent this procedure, 92% had iritis, 50% developed shallow anterior chamber, 33% had hyphaema and 22% had choroidal detachment.
- **Complications of Glaucoma Drainage Device**: Among 4 patients who underwent Ahmed Glaucoma Valve implantation, 75% had iritis, 25% had hyphaema and 25% had shallow anterior chamber during the follow up period of the study.
- **Complications of Anti VEGF:** Among 6 cases who received intravitreal anti VEGF, 50% developed transient increase in IOP following injection which resolved with medical treatment.
- **Complications of medical treatment:** In 23 patients who were on medical treatment alone, 73.90% did not have any significant complications while 26.08% had conjunctival hyperemia and another 26.08% had persistent pain.

These emphasise that surgical procedure had large number of complications that were also difficult to manage.

13. **Final visual outcome:** In our study, out of 92 patients, who presented to us with a poor vision of ≤6/60, only 26 improved to have better vision of > 6/60

after employing the above said modalities of treatment, while 66 patients persisted to have poor vision of ≤6/60.

This emphasises that neovascular glaucoma is refractory and is associated with poor visual prognosis. Other studies have also highlighted the poor visual outcomes in cases of neovascular glaucomas.[18][17]

14. **Final Intraocular pressure control:** In our study, among the 81 patients who presented with a very high IOP of >30mmHg, only 51 patients attained a good control with a final IOP of ≤30mmHg, while 30 patients had a persistent elevation of more than 30mmHg.

    This clearly shows that even after various modalities of treatment employed in a tertiary care centre, a significant portion of patients donot achieve IOP control highlighting the refractory nature of the disease and its poor prognosis.

CONCLUSION

- Neovascular glaucoma presents more commonly in advanced ages that is more than 60 years.
- Presentation is mostly in advanced stages, in stage 3 and 4 that is open angle and angle closure stages.
- The most common etiology is diabetic retinopathy and vascular occlusion. The pathogenesis of disease being neovascularisation in response to retinal ischemia. Hence all diabetic patients or patients with vessel occlusion should be carefully monitored and followed up for early detection of neovascularisation. Also, early treatment aimed at controlling the ischemic drive in high risk patients will prevent the development of neovascularisation.
- Visual outcomes and IOP control are poor in patients presenting in angle closure stage than open angle glaucoma stage, which again highlights the importance of careful screening of diabetic retinopathy and vascular occlusion patients for intraocular pressure, neovascularisation of iris and angles. This helps in identifying patients in early stages of the disease thereby, improving the prognosis.
- Various modalities of treatment are available, they have to be chosen according to etiology and stage of presentation. Angle closure patients have been proven to have poorer prognosis and hence early surgical intervention wherever possible has to be employed.
- Surgical intervention is required in most of the cases, but most of them are found to have increased complications and patients must be explainedabout the need for continuing treatment even after surgery and prolonged follow up.

- Neovascular glaucomas are generally associated with poor visual outcomes and present a therapeutic challenge.

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PROFORMA

CASE NUMBER:

1. Name: **Age / Sex:** **Mobile No:**

OP No. / Date:

Place:

2. Symptoms: (Duration)

Defective vision Pain Redness Photophobia

Any other symptoms:

3. Past History -

History of any intraocularsurgery / Trauma / Laser

4. Medical History –

Diabetes / Hypertension / IHD / Vaso Occlusive disease / Bronchial Asthma / Chronic disease / Steroid usage (long term)

5. Family History-

History of Diabetes / Hypertension / Ischemic Heart disease / Vein Occlusion diseases in the family

6. Ocular Examination:

Vision: (BCVA)

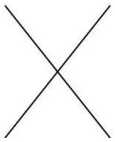
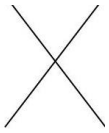
RE: **LE:**

Tension:

GAT/Rebound tonometry: RE: mmHg LE: mmHg

Slit Lamp Examination

	RE	LE
Lids		
Conjunctiva		
Cornea		
Anterior chamber: Depth		
Iris		
Pupil:		
Lens:		

Gonioscopy: Schaffer RE  **LE** 

Fundus : RE: media: **Disc and vessels:**

LE: media: **Disc and vessels:**

7. Other Investigations:

8. Diagnosis:

9.Treatment Plan:

Medical:Topical drugs / Lasers / Intravitreal AntiVEGF

Surgical: Filtering surgeries / Cyclo destructive procedures

10. Follow up: Monthly Visits

Visual acuity

Anterior segment

Fundus

IOP

Gonioscopy

KEY TO MASTER CHART

RE - RIGHT EYE, **LE** – LEFT EYE

GENDER:

M - MALE

F – FEMALE

VISION :

NOPL-NO PERCEPTION OF LIGHT

PL-PERCEPTION OF LIGHT

HM-HAND MOVEMENTS

CFCF-COUNTING FINGERS CLOSE TO FACE

IOP:INTRAOCULAR PRESSURE

CORNEA:

C – CLEAR

E – EDEMA

V – VASCULARISATION

IRIS:

CPN –COLOR PATTERN NORMAL

AP – ATROPHIC PATCHES

PS – POSTERIOR SYNECHIAE

SP – SECLUSIO PUPILLAE

OP – OCCLUSIO PUPILLAE

NVI – NEOVASCULARISATION OF IRIS

IB – IRIS BOMBE

ANTERIOR CHAMBER (AC):

ND – NORMAL DEPTH

S – SHALLOW

LENS:

C – CLEAR

IMC – IMMATURE CATARACT

MC – MATURE CATARACT

A – APHAKIA

PCIOL – POSTERIOR CHAMBER INTRAOCULAR LENS

ACIOL – ANTERIOR CHAMBER INTRAOCULAR LENS

FUNDUS:

NAD – NOTHING ABNORMAL DETECTED

NV – NO VIEW

GOA – GLAUCOMATOUS OPTIC ATROPHY

FSH – FLAME SHAPED HEMORRHAGE

CWS – COTTONWOOL SPOT

TT – TRAM TRACKING OF VESSEL

VS – VENOUS SHEATHING

DH – DOT HEMORRHAGE

BH – BLOT HEMORRHAGE

HE – HARD EXUDATE

DP - DISC PALLOR

NVD – NEOVASCULARISATION OF DISC

NVE – NEOVASCULARISATION ELSEWHERE

PRPM – PANRETINALPHOTOCOAGULATION MARKS

0.3-0.8 – CUP DISC RATIO

GONIOSCOPY:

1,2,3 – SHAFFER'S GRADING

CL –CLOSED

NVA-NEOVASCULARISATION OF ANGLE

NV-NO VIEW

ANTERIOR CHAMBER (A.C):

ND – NORMAL DEPTH

S – SHALLOW

DIAGNOSIS:

RNVG – RIGHT EYE NEOVASCULAR GLAUCOMA

LNVG – LEFT EYE NEOVASCULAR GLAUCOMA

ETIOLOGY:

PDR – PROLIFERATIVE DIABETIC RETINOPATHY

CRVO – CENTRAL RETINAL VEIN OCCLUSION

BRVO – BRANCH RETINAL VEIN OCCLUSION

CRAO – CENTRAL RETINAL ARTERY OCCLUSION

CU – CHRONIC UVEITIS

POAG – PRIMARY OPEN ANGLE GLAUCOMA

PACG – PRIMARY ANGLE CLOSURE GLAUCOMA

PXFG – PSEUDOEXFOLIATION GLAUCOMA

POST RD – POST RETINAL DETACHMENT SURGERY

UGH – UVEITIS GLAUCOMA HYPHAEMA SYNDROME

STAGE OF PRESENTATION:

OAG – OPEN ANGLE GLAUCOMA

ACG – ANGLE CLOSURE GLAUCOMA

AG – ABSOLUTE GLAUCOMA

TREATMENT:

PRP – PANRETINAL PHOTOCOAGULATION

TRAB + AM – TRABECULECTOMY WITH ANTIMETABOLITES

GDD – GLAUCOMA DRAINAGE DEVICE

AV- INTRAVITREAL BEVAZICUMAB INJECTION

IOLR +TRAB + AM – IOL REMOVAL WITH TRABECULECTOMY WITH ANTIMETABOLITES

M – ONLY MEDICAL TREATMENT

CYC – CYCLOCRYOTHERAPY

COMPLICATIONS:

I – IRITIS

HY – HYPHAEMA

H – VITREOUS HEMORRHAGE

S – SHALLOW ANTERIOR CHAMBER

CD – CHOROIDAL DETACHMENT

T – TRANSIENT RISE IN IOP

CH – CONJUCTIVAL HYPEREMIA

PP – PERSISTENT PAIN

S. No	Name	Age	Gender	Vision (RE)	Vision (LE)	IOP RE (mmHg)	IOP LE (mmHg)	Cornea (RE)	Cornea (LE)	AC (RE)	AC (LE)	Iris (RE)	Iris (LE)	Lens (RE)	Lens (LE)	Gonio (RE)	Gonio (LE)	Fundus (RE)	Fundus (LE)	Diagnosis	Etiology	Stage of Presentation	Treatment	Post treatment Vision	Post treatment IOP	Complic ations
1	Yogeswaran	70	M	HM+	6/18	42	18	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.4,NVE	DH,BH,HE	RNVG	PDR	ACG	TRAB+AM	4/60	34	I, HY
2	Ramjanhee	60	F	CFCF	6/9	51	19	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.4,NVE	NVD,NVE	RNVG	PDR	ACG	TRAB+AM	5/60	32	I, S
3	Malathi	33	F	6/18	HM+	12	42	C	E,V	ND	S	PS	PS,NVI	IMC	IMC	3	CL	NAD	0.8,DP	LNVG	CU	ACG	GDD	6/60	24	I, S
4	Meera Jul	34	F	6/9	PL+	12	42	C	E	ND	S	CPN	NVI, PS	C	MC	3	CL	NAD	0.8,DP	LNVG	CU	ACG	TRAB+AM	6/60	30	I, S
5	Manokar	57	M	6/6	No PL	18	32	C	E.V	ND	S	CPN	NVI, OP	C	IMC	3	CL	NAD	NV	LNVG	CU	AG	M	NO PL	30	-
6	Devi	55	F	PL+	4/60	40	28	E	E	S	S	NVI	PI	IMC	IMC	CL	CL	GOA	GOA	RNVG	PACG	ACG	TRAB+AM	5/60	38	I,HY
7	Nagappan	60	M	CFCF	6/18	40	18	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	O.5,NVD	O.4, NVE	RNVG	PDR	OAG	PRP	6/60	28	I,T
8	Muhamed	64	M	No PL	6/18	38	12	E,V	C	S	ND	NVI	CPN	IMC	IMC	CL	3	FSH,CWS,DP	NAD	RNVG	CRVO	AG	CYC	NO PL	4	-
9	Selvam	57	M	6/6	HM+	18	30	C	E	ND	ND	CPN	NVI,PS	C	IMC	3	NVA	NAD	0.5	LNVG	CU	OAG	M	4/60	28	-
10	Asmida Beevi	52	F	4/60	PL+	16	38	C	E	ND	S	CPN	NVI	IMC	IMC	3	CL	NAD	0.3,FSH,CWS,NVE	LNVG	CRVO	ACG	TRAB+AM	3/60	30	I,S,HY
11	Abdul Rahman	70	M	No PL	6/18	34	10	E,V	C	ND	ND	NVI	CPN	A	IMC	2	3	NAD	NAD	RNVG	CU	AG	M	NO PL	30	-
12	Venkatesan	60	M	6/18	CFCF	14	42	C	E	ND	ND	CPN	NVI	IMC	IMC	3	NVA	NAD	FSH,CWS,NVD	LNVG	CRVO	OAG	PRP	6/60	28	I
13	Seethaiyya	55	M	6/9	PL+	10	38	C	E,V	ND	ND	CPN	NVI	IMC	IMC	3	2	NAD	0.3,TT,VS,NVE	LNVG	CRAO	OAG	M	1/60	28	CH
14	Vassudevan	76	M	1/60	No PL	18	50	C	E,V	ND	ND	AP	NVI	PCIOL	PCIOL	3	CL	0.7	NV	LNVG	POAG	AG	CYC	NO PL	4	-
15	Vasanthamma	60	F	6/60	PL+	10	46	C	E	ND	S	CPN	NVI,IB	PCIOL	IMC	3	CL	NAD	0.7,DP	LNVG	CU	ACG	GDD	2/60	26	I
16	Nandhini	46	F	6/9	6/18	16	40	C	E	ND	ND	CPN	NVI	IMC	IMC	3	NVA	BH,HE	0.4,NVD	LNVG	PDR	OAG	PRP	6/9	22	-
17	Ramakrishnan	53	M	6/12	No PL	10	28	C	E,V	ND	ND	CPN	NVI	IMC	PCIOL	3	CL	0.3, NVD	NV	LNVG	PDR	AG	M	NO PL	30	-
18	Selvam	57	M	6/24	HM+	10	22	C	E	ND	ND	CPN	NVI	PCIOL	ACIOL	3	NVA	NAD	0.3	LNVG	UGH	OAG	LR+TRAB+A	6/60	28	I,HY
19	Janakiraman	69	M	½/60	No PL	18	42	C	E.V	ND	ND	CPN	NVI	IMC	IMC	2	NVA	0.8	0.7	LNVG	POAG	AG	CYC	NO PL	3	-
20	Kamala	75	F	6/18	PL+	20	40	C	E	ND	S	CPN	NVI	IMC	IMC	3	CL	0.3,NVE	0.3,NVD,NVE	LNVG	PDR	ACG	TRAB+AM	3/60	30	I,CD
21	Dayalan	58	M	HM+	1/60	48	10	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	0.3,NVD,NVE	0.3,HE,BH	RNVG	PDR	ACG	PRP	6/60	26	I
22	Jayakumar	51	M	HM	6/24	44	18	E	C	S	ND	NVI	CPN	PCIOL	IMC	CL	3	NV	0.4,PRPM	RNVG	PDR	ACG	M	3/60	34	PP
23	Soundarya	78	F	PL+	6/18	30	8	E	C	ND	ND	NVI,PS	AP	PCIOL	PCIOL	2	3	0.4,DP	0.3,DP	RNVG	CU	OAG	M	3/60	22	CH
24	Arunachalam	47	M	PL+	6/9	50	18	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	0.3,VS,FSH,NVI	NAD	RNVG	CRVO	ACG	TRAB+AM	3/60	40	I,HY
25	Jemes	66	M	HM+	6/9	40	18	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	0.3,NVD,NVE	DH,H,HE	RNVG	PDR	OAG	PRP	6/60	28	I,T
26	Abdul Malik	54	M	6/18	6/36	18	50	C	E	ND	S	CPN	NVI	IMC	IMC	3	CL	NVE	NVD,NVE	LNVG	PDR	ACG	PRP	6/18	24	-
27	Gandhi	61	M	HM+	6/9	28	10	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	2	FSH,VS,CWS	NAD	RNVG	BRVO	OAG	PRP	5/60	32	I,T
28	Pitchaikaran	65	F	3/60	PL+	14	40	C	E	ND	ND	CPN	NVI	IMC	IMC	2	2,NVA	0.7	0.8,DP	LNVG	POAG	OAG	M	6/60	30	-
29	Andal	50	F	PL+	6/60	42	15	E	C	S	ND	NVI,OP	CPN	IMC	IMC	CL	3	0.8,DP	NAD	RNVG	CU	ACG	TRAB+AM	1/60	32	I,S,CD
30	Azhagamma	55	F	3/60	6/24	42	10	E	C	ND	ND	NVI	CPN	IMC	IMC	2	3	0.3,NVE	DH,BH,HE	RNVG	PDR	OAG	PRP	6/36	26	I
31	Mariammal	51	F	PL+	6/60	48	14	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	0.3,NVE	NVE	RNVG	PDR	OAG	PRP	1/60	32	I,T,H
32	Shed Hilal	60	M	6/60	3/60	58	22	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	2	0.3,NVE	DH,BH,HE	RNVG	PDR	OAG	PRP	6/36	28	I,T
33	Vanam	70	F	6/60	6/12	26	12	E	C	ND	ND	NVI	CPN	IMC	IMC	2	3	0.5,NVE	0.3,NVE	RNVG	PDR	OAG	PRP	6/36	26	I
34	Thomas	65	M	3/60	6/18	50	12	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	0.3,NVD,NVE	0.3,NVE	RNVG	PDR	OAG	PRP	6/36	24	I
35	Parvathi	80	F	1/60	1/60	53	16	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	0.5,NVD,NVE	0.3,NVE	RNVG	PDR	OAG	PRP	6/60	32	I,T
36	Eganathan	60	M	5/60	6/36	57	20	E	C	ND	ND	NVI	CPN	IMC	PCIOL	NVA	3	0.3,NVE	O.3,NVE	RNVG	PDR	OAG	PRP	6/36	24	I
37	Jayasree	63	F	6/60	6/24	44	14	E	C	ND	ND	NVI	CPN	IMC	PCIOL	NVA	3	0.3,NVD,NVE	0.3,NVE	RNVG	PDR	OAG	PRP	6/24	24	I
38	Sakila	50	F	6/60	6/9	50	20	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.5,NVE	DH,BH,HE	RNVG	PDR	ACG	TRAB+AM	6/24	32	I,S
39	Govindan	64	M	6/60	2/60	10	42	C	E	ND	S	CPN	NVI	IMC	IMC	3	CL	0.3,DH,BH	0.5,NVE	LNVG	PDR	ACG	TRAB+AM	6/36	26	I,S
40	Balakrishnan	57	M	6/60	6/9	34	22	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.4,NVE	BH,HE	RNVG	PDR	ACG	TRAB+AM	6/36	26	I,S

S. No	Name	Age	Gender	Vision (RE)	Vision (LE)	IOP RE (mmHg)	IOP LE (mmHg)	Cornea (RE)	Cornea (LE)	AC (RE)	AC (LE)	Iris (RE)	Iris (LE)	Lens (RE)	Lens (LE)	Gonio (RE)	Gonio (LE)	Fundus (RE)	Fundus (LE)	Diagnosis	Etiology	Stage of Presentation	Treatment	Post treatment Vision	Post treatment IOP	Complic ations
41	Lakshmi Kandaiya	64	M	6/60	6/36	40	18	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	0.6,NVE,DP	BH,HE	RNVG	PDR	ACG	TRAB+AM	6/24	24	I,S
42	Iyyappan	78	M	2/60	6/60	41	20	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	0.6,NVE,DP	0.3,NVE	RNVG	PDR	ACG	TRAB+AM	1/60	30	I,CD
43	Pachaiyyappan	43	M	6/24	1/60	18	50	C	E	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	0.3,NVE	0.5,NVE	LNVG	PDR	ACG	GDD	3/60	28	I
44	Govindhasamy	70	M	PL+	6/24	43	12	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.6,NVE,DP	0.3,NVD.NVE	RNVG	PDR	ACG	TRAB+AM	1/60	38	I,S,HY
45	Jayaraman	65	M	6/60	HM+	22	50	C	E	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	0.3,NVE	0.5,NVE	LNVG	PDR	ACG	TRAB+AM	3/60	38	I,S,HY
46	Panchacharam	73	M	2/60	6/36	44	20	E	C	S	ND	NVI	CPN	IMC	IMC	CL	2	0.5,NVD,NVE	0.3,NVE	RNVG	PDR	ACG	PRP	6/36	24	I
47	Kumari	63	F	6/18	2/60	16	36	C	E	ND	S	CPN	NVI	IMC	IMC	2	CL	0.3,BH.HE	0.5,NVD,NVE	LNVG	PDR	ACG	PRP	6/36	32	I
48	Samini	61	F	1/60	4/60	46	12	E	C	S	ND	NVI	CPN	IMC	IMC	CL	2	0.5,NVD,NVE	0.3,NVE	RNVG	PDR	ACG	TRAB+AM	4/60	32	I,S,HY
49	Ramachandran	63	M	No PL	6/36	46	22	E.V	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	NV	NAD	RNVG	CRVO	AG	M	NO PL	30	-
50	Venkatesan	39	M	1/60	3/60	54	12	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	FSH,CWS,DP	NAD	RNVG	CRVO	ACG	TRAB+AM	3/60	42	I,HY
51	Saraswathi	69	F	3/60	HM+	18	44	C	E,V	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	0.3,NVE	NV	LNVG	PDR	ACG	M	1/60	36	PP
52	Semari	52	F	HM +	6/12	44	12	E	C	S	ND	NVI	CPN	PCIOL	IMC	CL	2	0.3,NVE	DH,BH,HE	RNVG	PDR	ACG	TRAB+AM	2/60	34	I,S,HY
53	Abdul Sherif	51	M	PL +	6/60	48	13	E	C	S	ND	NVI	CPN	PCIOL	IMC	CL	2	0.6,NVD,NVE	0.3,NVE	RNVG	PDR	ACG	TRAB+AM	1/60	36	I,S,HY
54	Rajana	56	M	6/12 P	6/24	14	30	C	E	ND	S	CPN	NVI	IMC	IMC	2	CL	0.3,NVE	0.3,NVE	LNVG	PDR	ACG	PRP	6/18	22	-
55	Mariammal	51	F	6/36	6/12	32	20	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.3,NVE	0.3,NVE	RNVG	PDR	ACG	PRP	6/18	18	I
56	Devaki	55	F	6/36	6/6	28	12	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.3,NVD,NVE	0.3,NVE	RNVG	PDR	ACG	PRP	6/24	18	I
57	Sariasti	36	F	6/36	6/12	30	16	E	C	S	ND	NVI	CPN	IMC	IMC	CL	2	0.3,NVD,NVE	0.3,NVE	RNVG	PDR	ACG	PRP	6/24	22	I
58	V. Raman	57	M	6/60	6/12	38	20	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.3,NVE	0.3,NVE	RNVG	PDR	ACG	PRP	6/24	22	I,T
59	Mangalam	52	M	2/60	6/9	30	20	E	C	ND	ND	NVI	CPN	IMC	IMC	2	2	FSH,CWS	NAD	RNVG	CRVO	OAG	AV	5/60	18	-
60	Chandra	62	F	6/36	6/12	28	12	E	C	ND	ND	NVI	CPN	IMC	IMC	2	2	FSH,CWS	NAD	RNVG	CRVO	OAG	AV	6/18	18	-
61	Sivagamy	66	F	3/60	3/60	26	10	E	C	ND	ND	NVI	CPN	IMC	IMC	2	2	FSH,CWS	NAD	RNVG	CRVO	OAG	AV	6/60	22	T
62	Chandran	61	M	2/60	3/60	14	28	C	E	ND	ND	CPN	NVI	IMC	IMC	2	2	NAD	FSH,CWS	LNVG	CRVO	OAG	AV	6/36	16	-
63	Munusamy	35	M	6/24	6/60	16	26	C	E	ND	ND	CPN	NVI	IMC	IMC	2	NVA	NAD	FSH,CWS	LNVG	CRVO	OAG	AV	6/36	22	T
64	Geetha	60	F	6/12	1/60	18	32	C	E	ND	ND	CPN	NVI	IMC	IMC	2	NVA	NAD	FSH,CWS	LNVG	CRVO	OAG	AV	5/60	22	T
65	Varadhan	65	M	HM+	6/60	36	20	E	C	ND	ND	NVI	CPN	PCIOL	PCIOL	NVA	3	FSH,CWS,NVE	NAD	RNVG	CRVO	OAG	PRP	4/60	30	I,T
66	Mangavaram	50	F	6/6	2/60	12	28	C	E	ND	ND	CPN	NVI	PCIOL	PCIOL	3	NVA	NAD	FSH,CWS,NVD	LNVG	CRVO	OAG	PRP	4/60	34	I,T
67	Muhamed Unis	64	M	5/60	6/12	42	12	E	C	ND	ND	NVI	CPN	IMC	IMC	2	2	FSH,CWS	NAD	RNVG	BRVO	OAG	M	6/36	30	-
68	Asir Deva Sagayan	48	M	2/60	6/18	52	10	E	C	ND	ND	NVI	CPN	PCIOL	PCIOL	2	2	FSH,CWS	NAD	RNVG	CRVO	OAG	M	5/60	32	-
69	Gopal	67	M	3/60	6/12 P	40	18	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	2	FSH,CWS	NAD	RNVG	BRVO	OAG	M	6/36	30	-
70	Shantha	60	F	6/24	2/60	12	60	C	E	ND	ND	CPN	NVI	PCIOL	PCIOL	3	NVA	NAD	FSH,CWS,NVD	LNVG	BRVO	OAG	PRP	6/36	26	I
71	Padma	66	F	4/60	6/60	38	17	E	C	ND	ND	NVI	CPN	PCIOL	PCIOL	NVA	3	H,CWS,NVE,NV	NAD	RNVG	BRVO	OAG	PRP	6/24	24	I,T
72	Elumalai	68	M	6/18	CFCF	14	38	C	E	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	NAD	FSH,CWS	LNVG	CRVO	ACG	TRAB+AM	2/60	32	I,S,HY
73	Subramani	47	M	5/60	6/6	42	16	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	CWS,TT,VS	NAD	RNVG	CRAO	ACG	M	6/60	32	-
74	Elumalai	63	M	PL+	6/24 P	38	12	E	C	S	ND	NVI	CPN	IMC	IMC	CL	2	FSH,CWS	NAD	RNVG	CRVO	ACG	TRAB+AM	5/60	30	I,S
75	Elumalai	65	M	PL+	2/60	50	22	E	C	S	ND	NVI	CPN	IMC	IMC	CL	2	FSH,CWS	NAD	RNVG	CRVO	ACG	TRAB+AM	6/60	30	I,S
76	Ragini	60	F	5/60	PL+	10	60	C	E	ND	S	CPN	NVI	IMC	IMC	2	CL	NAD	FSH,CWS	LNVG	CRVO	ACG	TRAB+AM	2/60	32	I,S,CD
77	Murugesan	42	M	PL+	6/9	54	14	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	FSH,CWS	NAD	RNVG	CRVO	ACG	TRAB+AM	2/60	30	I,S,CD
78	Parimal	48	M	HM+	6/6 P	30	14	E	C	S	ND	NVI	CPN	IMC	IMC	CL	3	FSH,CWS	NAD	RNVG	BRVO	ACG	TRAB+AM	6/60	24	I,S
79	Suseela	60	F	1/60	6/18	40	16	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	FSH,CWS	NAD	RNVG	CRVO	ACG	TRAB+AM	6/60	30	I,S
80	Mallika	58	F	PL+	6/12	52	20	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	FSH,CWS	NAD	RNVG	CRVO	ACG	GDD	2/60	28	I, HY

S. No	Name	Age	Gender	Vision (RE)	Vision (LE)	IOP RE (mmHg)	IOP LE (mmHg)	Cornea (RE)	Cornea (LE)	AC (RE)	AC (LE)	Iris (RE)	Iris (LE)	Lens (RE)	Lens (LE)	Gonio (RE)	Gonio (LE)	Fundus (RE)	Fundus (LE)	Diagnosis	Etiology	Stage of Presentation	Treatment	Post treatment Vision	Post treatment IOP	Complications
81	Renu Gopal	42	F	HM+	6/18	34	12	E	C	ND	ND	NVI	CPN	IMC	IMC	2	3	0.6,DP	0.7	RNVG	POAG	OAG	TRAB+AM	5/60	24	I,CD
82	Amir Basha	65	M	6/9	2/60	14	40	C	E	ND	ND	CPN	NVI	IMC	IMC	2	2	0.6	0.8	LNVG	POAG	OAG	TRAB+AM	6/60	24	I
83	Banumathi	62	F	6/36	6/12	36	12	E	C	ND	ND	NVI	CPN	IMC	IMC	3	3	0.8	0.8	RNVG	POAG	OAG	TRAB+AM	6/24	26	I
84	Mathiazhgi	66	F	3/60	3/60	36	10	E	C	ND	ND	NVI	CPN	IMC	IMC	NVA	3	0.9	0.7	RNVG	POAG	OAG	TRAB+AM	6/36	24	I,S
85	Pichandi	61	M	2/60	3/60	14	32	C	E	ND	ND	CPN	NVI	IMC	IMC	3	NVA	0.6	0.8	LNVG	POAG	OAG	TRAB+AM	6/36	28	I,S
86	Periyadurai	78	M	6/24	2/60	16	26	C	E	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	0.6	0.8	LNVG	PACG	ACG	M	6/60	22	-
87	Ponnamal	60	F	6/12	1/60	18	32	C	E	ND	S	CPN	NVI	PCIOL	PCIOL	3	CL	0.7	0.8	LNVG	PACG	ACG	M	6/36	22	-
88	venkatachalam	65	M	HM+	6/60	36	20	E	C	S	ND	NVI	CPN	PCIOL	PCIOL	CL	3	0.9	0.7	RNVG	PACG	ACG	M	6/36	26	-
89	Manimegalai	50	F	6/36	2/60	12	28	C	E	ND	ND	PXF	PXF,NVI	IMC	IMC	2	NVA	0.7	0.9	LNVG	PXFG	OAG	M	6/60	24	-
90	Akram khan	64	M	5/60	6/12	40	12	E	C	ND	ND	PXF,NVI	PXF	IMC	IMC	2	2	0.8	0.6	RNVG	PXFG	OAG	M	6/36	28	-
91	Sivakumar	48	M	2/60	6/18	40	10	E	C	ND	ND	NVI,PS	PS	IMC	IMC	NVA	3	0.7	NAD	RNVG	CU	OAG	TRAB+AM	6/36	26	I,HY
92	Palani	67	M	3/60	6/12 P	30	18	E	C	ND	ND	NVI,PS	PS	IMC	IMC	NVA	3	0.5	NAD	RNVG	CU	OAG	TRAB+AM	6/36	22	I,HY
93	Pownammal	60	F	6/24	2/60	12	50	C	E	ND	S	PS	OP,NVI	IMC	IMC	2	CL	NAD	0.8,DP	LNVG	CU	ACG	TRAB+AM	5/60	30	I,S
94	Murugan	66	M	4/60	6/60	38	17	E	C	S	ND	NVI	PS	IMC	IMC	CL	2	DP,0.8	NAD	RNVG	CU	ACG	TRAB+AM	6/60	30	I,S
95	Venkatesan	68	M	6/18	CFCF	14	40	C	E	S	S	CPN	PS,NVI	IMC	IMC	CL	CL	NAD	0.6	LNVG	CU	ACG	TRAB+AM	3/60	28	I,S,CD
96	Sivasndar	47	M	HM+	6/24	42	16	E	C	S	ND	PS,NVI	CPN	IMC	IMC	CL	2	NV	NAD	RNVG	CU	ACG	M	3/60	36	CH,PP
97	Arunagiri	63	M	PL+	6/24 P	38	12	E	C	S	ND	SP,NVI	CPN	IMC	IMC	CL	2	NV	NAD	RNVG	CU	ACG	M	3/60	34	CH,PP
98	Mannangatti	65	M	PL+	2/60	52	22	E,V	C	S	ND	SP,NVI	PS	IMC	IMC	CL	2	NV	NAD	RNVG	CU	ACG	M	2/60	38	CH,PP
99	Jeyaraman	60	M	5/60	PL+	10	46	C	E,V	ND	S	PS	OP,NVI	IMC	IMC	2	CL	NAD	NV	LNVG	CU	ACG	M	2/60	32	CH,PP
100	Deivanai	50	F	No PL	6/36	42	14	E.V	C	S	ND	NVI	CPN	PCIOL	IMC	CL, SO	2	NV	0.3	RNVG	POST RD	AG	CYC	NO PL	2	-